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Dedication

Allah the all merciful, I beg thee to accept this effort for the soul of my father
He was your gift for me

Do you find yourself confused while reading an ECG?

Tired of getting through various text books?

Need an easy way to recall basis of ECG?

The answer to all your ECG problems lies in this simple yet comprehensive book, Starting from the basic cardiac physiology ending with the various abnormalities encountered, you will find yourself easily an expert in interpreting and extracting wealthy information from the ECG.

The excellence of this book does not only lie in its simple read, understand and recall way, but in the fact that it was written with the love of giving and passing knowledge. Graciously accept this book, for as we can only give ourselves by giving away to others.

Mahmoud Ameen
M.B.B.C.H.
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Introduction

The electrocardiogram (ECG or EKG) is a special graph that represents the electrical activity of the heart from one instant to the next. Thus, the ECG provides a time-voltage chart of the heartbeat. For many patients, this test is a key component of clinical diagnosis and management in both inpatient and outpatient settings. The device used to obtain and display the conventional ECG is called the electrocardiograph, or ECG machine. It records cardiac electrical currents (voltages or potentials) by means of conductive electrodes selectively positioned on the surface of the body.

This book is devoted to explaining the basis of the normal ECG and then examining the major conditions that cause abnormal depolarization (P and QRS) and repolarization (ST-T and U) patterns.

Why is the ECG so clinically useful ?

The ECG is one of the most versatile and inexpensive of clinical tests. Its utility derives from careful clinical and experimental studies over more than a century showing the following:

- It is the essential initial clinical test for diagnosing dangerous cardiac electrical disturbances related to conduction abnormalities in the AV junction and bundle branch system and to brady- and tachyarrhythmias.
- It often provides immediately available information about clinically important mechanical and metabolic problems, not just about primary abnormalities of electrical function. Examples include myocardial ischemia/infarction, electrolyte disorders, and drug toxicity, as well as hypertrophy and other types of chamber overload.
- It may provide clues that allow you to forecast preventable catastrophes. A good example is a very long QT(U) pattern preceding sudden cardiac arrest due to torsades de pointes.

Physiological anatomy of the heart :

The heart is a hollow muscular pump situated in the left side of the thoracic cavity partly behind the sternum, consisting of 4 chambers : 2 atria and 2 ventricles.

The heart is covered externally by **epicardium** (which is the visceral layer of the pericardial sac). The inside cavity of the heart lined by endothelial layer called the **endocardium**. An intermediate muscular layer lying in between the epicardium & endocardium known as the **myocardium**.

Physiology of Cardiac Muscle :

The heart is composed of three major types of cardiac muscle: atrial muscle, ventricular muscle, and specialized excitatory and conductive muscle fibers.

The atrial and ventricular types of muscle contract in much the same way as skeletal muscle, except that the duration of contraction is much longer. Conversely, the specialized excitatory and conductive fibers contract only feebly because they contain few contractile fibrils; instead, they exhibit either automatic rhythmical electrical discharge in the form of action potentials or conduction of the action potentials through the heart, providing an excitatory system that controls the rhythmical beating of the heart.

The cardiac muscle has certain special properties which are :

1. **Rhythmicity:** ability of the heart to beat regularly at constant rate.
2. **Contractility:** ability of the heart to contract and push blood into circulation.
3. **Excitability:** ability of the cardiac muscle to respond to an adequate stimulus contraction.
4. **Conductivity:** ability of the cardiac muscle to conduct excitation wave from one part of the heart to another.

In EKG study we are concerned with study of **Rhythmicity** and **conductivity** of the cardiac muscle.

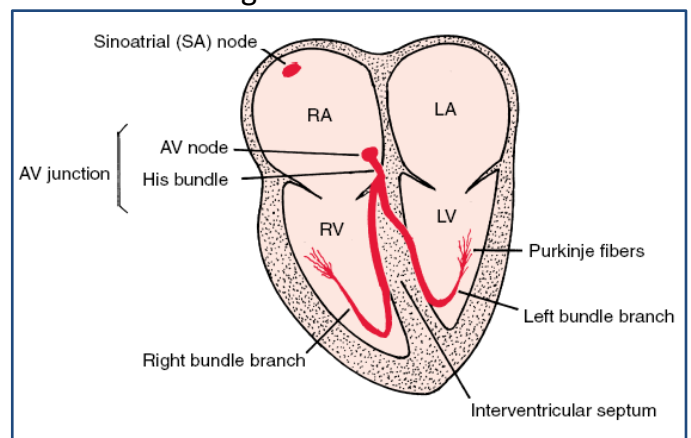
we will review a few simple principles of the heart's electrical properties. The central function of the heart is to contract rhythmically and pump blood to the lungs for oxygenation and then to pump this oxygen-enriched blood into the general (systemic) circulation. The signal for cardiac contraction is the spread of electrical currents through the heart muscle. These currents are produced both by *pacemaker cells* and *specialized conduction tissue* within the heart and by the working heart muscle itself.

Pacemaker cells are like tiny clocks (technically called oscillators) that repetitively generate electrical stimuli. The other heart cells, both specialized conduction tissue and working heart muscle, are like cables that transmit these electrical signals.

Electrical Activation of the Heart :

In simplest terms, therefore, the heart can be thought of as an electrically timed pump. The electrical "wiring" is outlined in Figure.

Normally, the signal for heartbeat initiation starts in the sinus or **sinoatrial (SA) node**. This node is located in the right atrium near the opening of the superior vena cava.



The SA node is a small collection of specialized cells capable of automatically generating an electrical stimulus (spark-like signal) and functions as the normal pacemaker of the heart. From the sinus node, this stimulus spreads first through **the right atrium** and then into the **left atrium**. Electrical stimulation of the right and left atria signals the atria to contract and pump blood simultaneously through the tricuspid and mitral valves into the right and left ventricles.

The electrical stimulus then reaches specialized conduction tissues in the **atrioventricular (AV) junction**. The AV junction, which acts as an electrical “relay” connecting the atria and ventricles, is located at the base of the interatrial septum and extends into the interventricular septum. The upper (proximal) part of the AV junction is the AV node. (In some texts, the terms AV node and AV junction are used synonymously.) The lower (distal) part of the AV junction is called the **bundle of His**. The bundle of His then divides into two main branches: **the right bundle branch**, which distributes the stimulus to the right ventricle, and the **left bundle branch**, which distributes the stimulus to the left ventricle.

The electrical signal then spreads simultaneously down the left and right bundle branches into the ventricular myocardium (ventricular muscle) by way of specialized conducting cells called **Purkinje fibers** located in the subendocardial layer (inside rim) of the ventricles. From the final branches of the Purkinje fibers, the electrical signal spreads through myocardial muscle toward the epicardium (outer rim).

The His bundle, its branches, and their subdivisions are referred to collectively as His-Purkinje system. Normally, the AV node and His-Purkinje system form the only electrical connection between the atria and the ventricles (unless a bypass tract is present). Disruption of conduction over these structures will produce AV heart block.

Just as the spread of electrical stimuli through the atria leads to atrial contraction, so the spread of stimuli through the ventricles leads to ventricular contraction, with pumping of blood to the lungs and into the general circulation. The initiation of cardiac contraction by electrical stimulation is referred to as electromechanical coupling. A key part of this contractile mechanism is the release of calcium ions inside the atrial and ventricular heart muscle cells, which is triggered by the spread of electrical activation. This process links electrical and mechanical function.

The ECG is capable of recording only relatively large currents produced by the mass of working (pumping) heart muscle. The much smaller amplitude signals generated by the sinus node and AV node are invisible with clinical recordings.

Depolarization of the His bundle area can only be recorded from inside the heart during specialized cardiac electrophysiologic (EP) studies.

Heart has two types of action

- **Mechanical:** Contraction & relaxation
- **Electrical:** Depolarization & repolarization

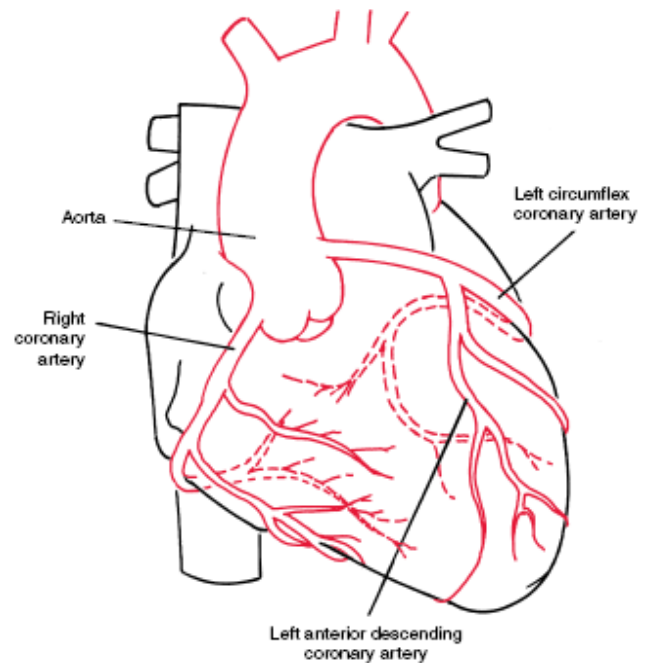
Blood supply of the heart through the coronary arteries

Anatomy of the coronary arteries

The left Coronary artery:

It arises from the left sinus of Valsalva and passes forwards & to the left in the atrioventricular groove for a short distance and then divides into two branches:

1. **The left anterior descending artery:** it passes downwards in the anterior interventricular groove to the apex of the heart & then turns backwards to anastomse with the posterior descending artery.
2. **The circumflex artery:** it continues its course in the left atrioventricular groove to anastomse with the right coronary. It gives several obtuse marginal branches.



The right Coronary artery:

It arises from the (right sinus) of Valsalva and runs in the right atrioventricular groove to the posterior surface of the heart to anastomse with circumflex artery.

In the back of the heart it gives the (posterior descending artery which runs downwards, in the posterior interventricular groove, to anastomose with the anterior descending artery.

Pattern of coronary supply

- **Balanced circulation:**

The left coronary artery supplies **left atrium**, **left ventricle** & **anterior part of the interventricular septum**.

While the right coronary artery supplies **right atrium**, **right ventricle** & **posterior part of the interventricular septum**.

- **Right dominance:**

The right coronary supplies also the posterior part of the left ventricle.

- **Left dominance:**

The left coronary supplies also the posterior part of the septum & the posterior wall of the right ventricle.

Normal ECG

Cardiac Electrical Activity

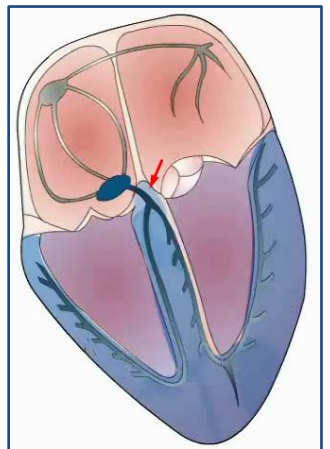
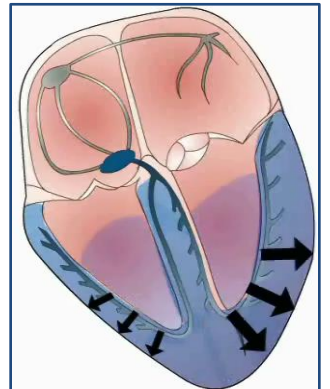
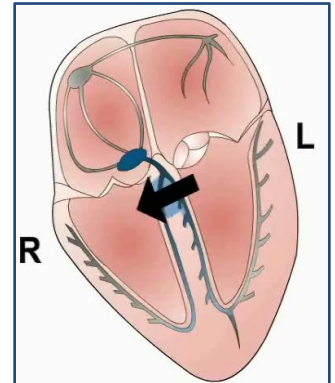
During each cardiac cycle, the atria contract in the diastole to fill the ventricles, while the ventricle contract during systole to supply blood to the lungs and systemic circulation. Contraction of the atria and ventricles is tightly coordinated by wave of depolarization spreading through the muscular wall of this chambers.

The interventricular septum is the first part of the ventricular muscle mass to be depolarized and it does so by movement of current across the septum from the left to the right bundle branch . we will see later this early left to right movement of current in the septum is crucial to understanding several important ECG abnormalities.

After septal depolarization, the depolarizing wave begins to spread rapidly to the bulk of the left and right ventricles.

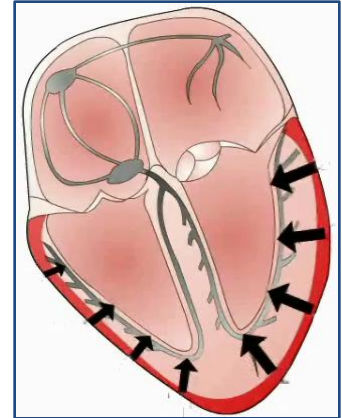
The ventricular depolarization spreads from terminal fibers of the conducting system outwards(from the endocardium towards the epicardial surface of the heart) and also back along the ventricular wall to the atrioventricular groove.

The final piece of muscle to be depolarized is the upper part of the interventricular septum



Cardiac repolarization is not truly propagated between cells, however cardiac myocytes repolarize at different rates depending on their anatomical locations within the heart. Within the ventricular wall there is a gradient in the rate of repolarization. Cells in the epicardial region has the fastest rate of repolarization and repolarize first following ventricular contraction. The rate of repolarization slows progressively as we move from the epicardium towards the endocardium.

We will see later this retrograde spread of ventricular repolarization is important in understanding the normal ECG reader



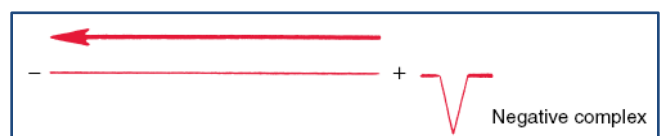
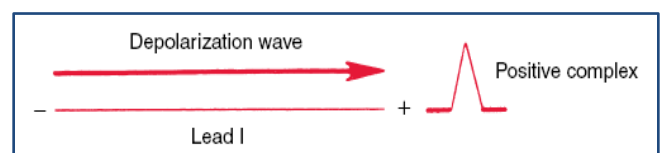
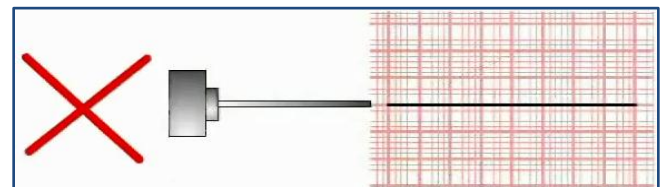
ECG Generation

Now, we are going to explain how the electric events are analyzed by the ECG leads to produce the waves. The leads of the ECG machine detect the movement of the cardiac depolarization and repolarization waves as they spread to the atria and ventricles.

Leads cables of detecting electric signals are placed on the patient body, and the different lead position record the flow of current through the heart from different respective. In this way the ECG recording can give information about these processes affecting different anatomical regions of the organ.

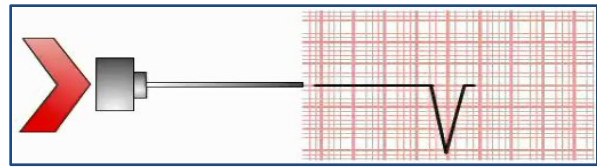
We need to understand how the individual ECG leads. Analysis and records the cardiac current.

- In any ECG lead, a flat line is recorded when no current is flowing >> iso-electric line.
- The depolarizing current moving towards the lead produces a deflection on the ECG paper above the iso-electric line (a positive deflection).
- The depolarizing current moving away from the lead produces a deflection but below the iso-electric line (a negative deflection)

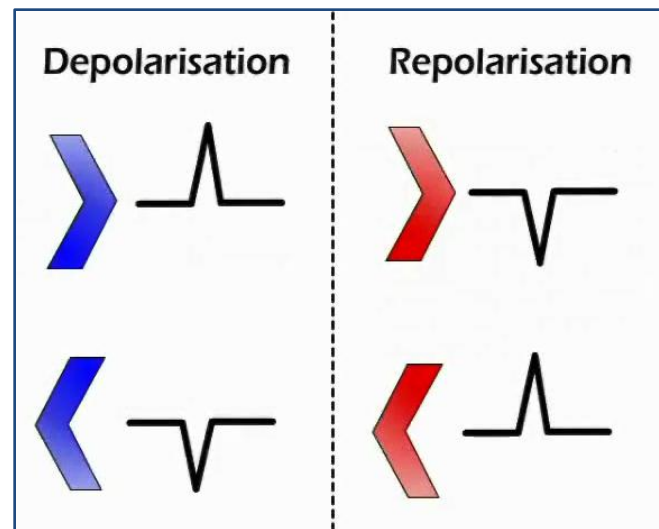


In contrast, repolarizing current has the opposite polarity to depolarizing current, Therefore :

- Repolarizing current moving toward the lead produces a negative deflection on the paper.
- While, repolarizing current moving away from the lead produces a positive deflection.

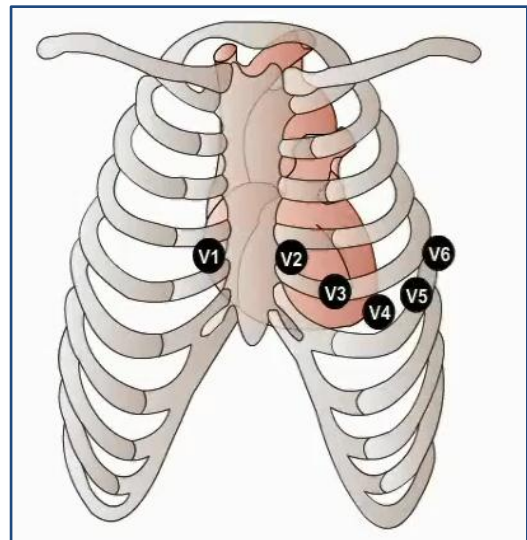


As the depolarization and repolarization waves spread over the normal heart in a well defined pattern. This means that, if we know the position of the ECG lead relatively to the heart, we can predict the form of the readers if recorded.

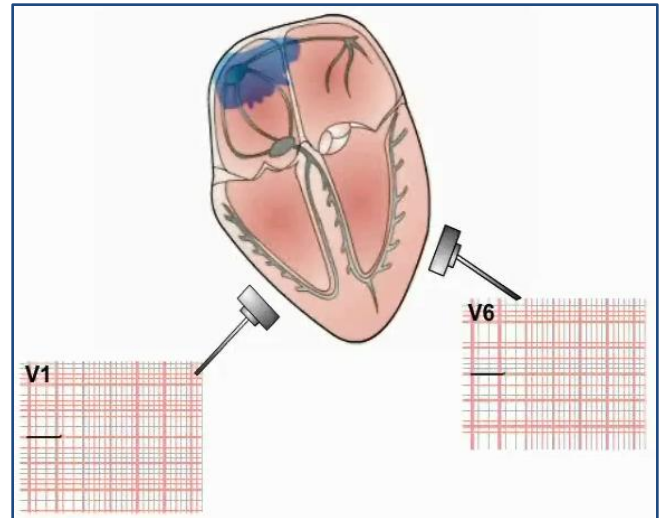


Let's see how this work on 2 of 6 (Chest leads V_1 to V_6)

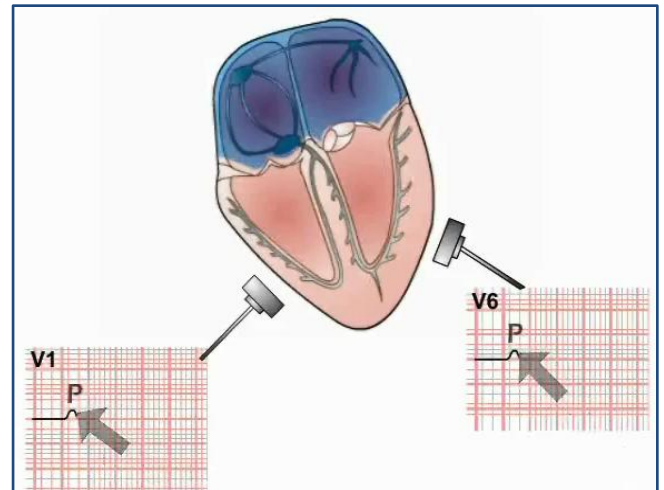
- Lead V_1 is placed on the anterior surface of the patient's chest in the 4th right intercostals space to the right of the sternum, and therefore to the right of the bulk of the ventricles.
- In contrast, lead V_6 is placed on the patient's chest in the 5th intercostals space mid-axillary line, and looks at the heart from the left of the ventricles.



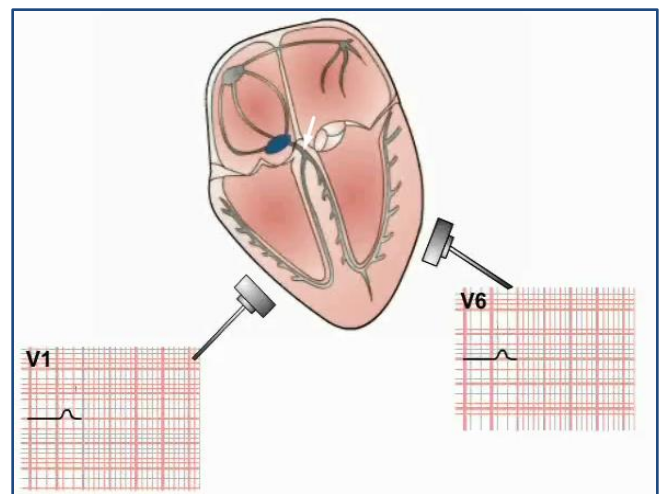
In each cardiac cycle atrial contraction is associated with a wave of depolarization spreading over the chambers. As the atria settled back in the chest cavity, this wave of depolarization is not only spreading downwards and to the left from the SA node, but also outwards to the front of the chest, and therefore to the chest leads.



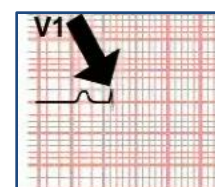
As depolarizing current moves towards the leads it produces a positive deflection on the ECG paper. This is the P wave (atrial depolarization)



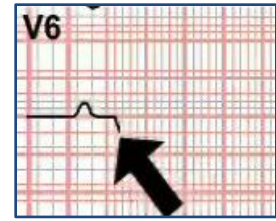
After a short delay, in which no current is flowing, the AV node allows the depolarization signals to travel into the ventricles. As seen, mid zone of the interventricular septum is the first piece of the ventricular muscle to depolarize and it does so by signals spreading across the septum from the left towards the right bundle branch.



These early depolarization signals move towards V₁ and therefore produce a positive deflection on the ECG paper in recording from this lead.



However, this septal current is moving away from lead V₆ producing an initial negative deflection in this lead.



As the septum continues to depolarize, the depolarization wave spreads out over the muscle mass of the ventricle.

To understand what happens next records, it is important to realize that the magnitude of the electric signal generated by the depolarizing muscle is directly proportional to the mass of this muscle.

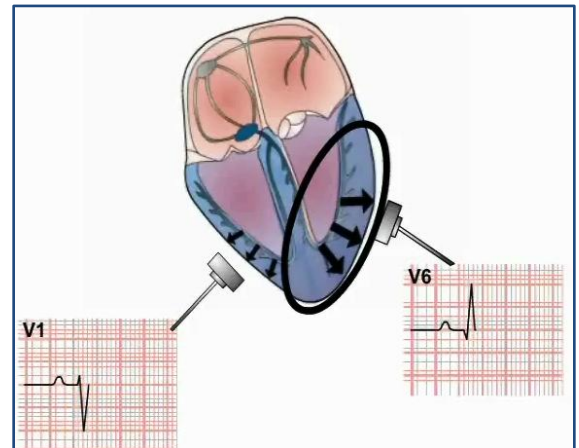
What is meant by that ???

The more muscle fibers present, the more electric signals generated and the more signals detected by the ECG machine.

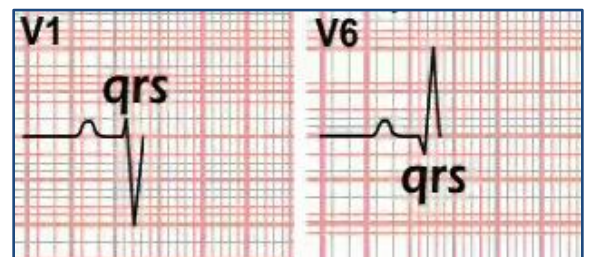
the left ventricle has a much greater muscle than the right, so, dominate the electric signals of the ventricular depolarization in all leads.

Therefore, as the wave of electrical activity reaches the main muscle mass of the ventricles, the left ventricular signals overwhelm all other signals. The wave in V₁ (the deflection) become negative

In contrast, this signals is moving towards V₆, producing a strong positive deflection



The flow of depolarizing current along the ventricles is recorded as QRS complex



When ventricular depolarization is completed, there is a brief period when no current is flowing and recording returns to the iso-electric line. This period ends with the onset of ventricular repolarization.

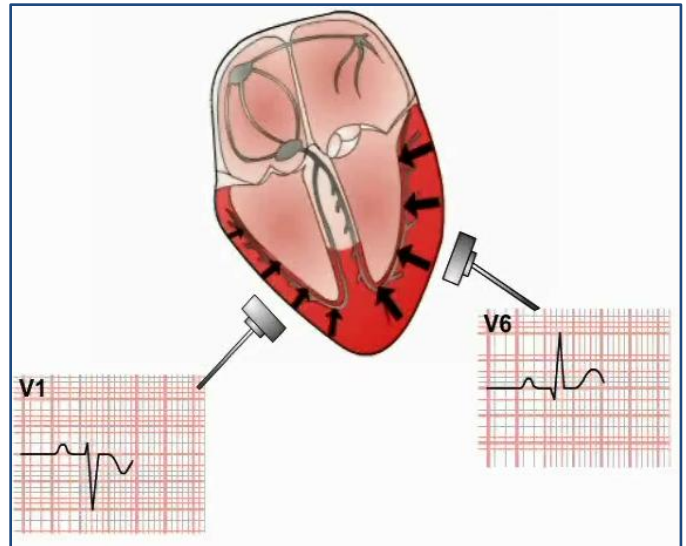
Remember repolarizing current has the opposite polarity to the depolarization wave, and therefore, when it is moving towards the lead it produces a negative deflection on ECG paper and positive deflection when moving away from a lead.

the deflection produced on an ECG by ventricular repolarization is dominated by the signals from the left ventricles

as this repolarizing current is moving towards V_1 >> the deflection produced >> is negative in this lead.

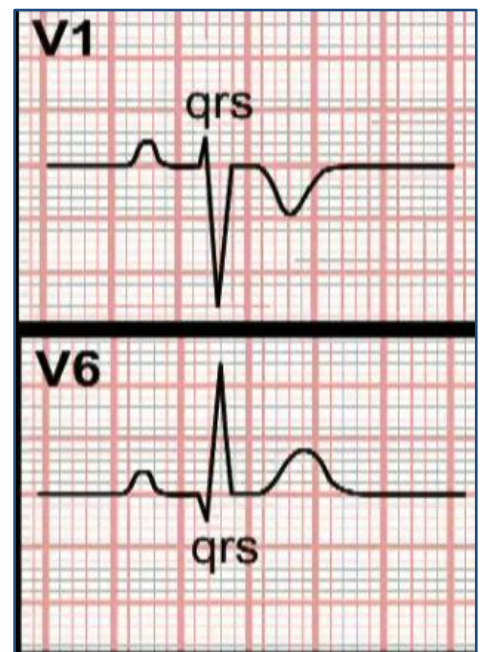
In contrast, this repolarizing signals is moving away from V_6 , producing a positive deflection

The deflection produced by a ventricular repolarization is termed a T wave



ECG Nomenclature

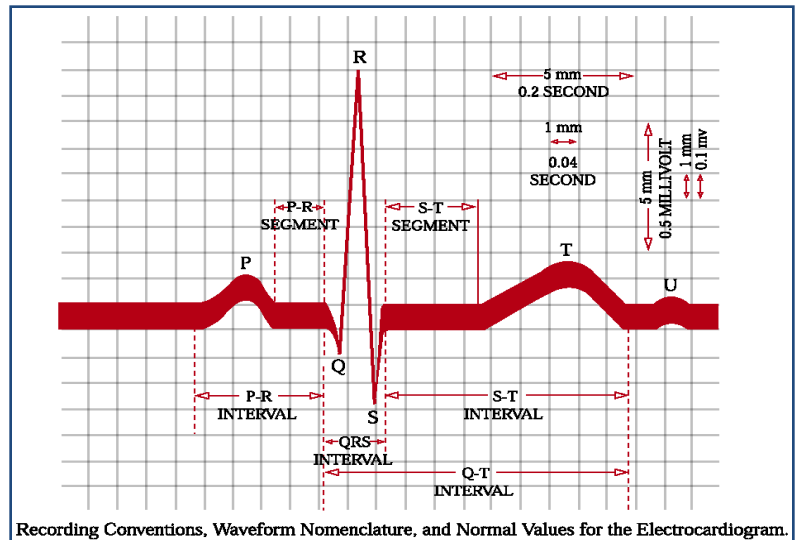
We need to make sure, that you are familiar with the nomenclature of the ECG waves, remember the deflection produced by the atrial depolarization is termed a **P** wave, while ventricular depolarization produces the **QRS** complex. The nomenclature of the QRS complex can cause some confusion. Within the QRS complex any positive deflection that is a deflection above the iso-electric line is termed a **R** wave. Any negative deflection which follows R wave is termed a **S** wave. However, if the first deflection of the QRS complex is negative, this deflection is termed a **Q** wave. This is important a Q wave can only exist if, and only if, the first deflection of the QRS complex is negative. And the negative deflection following a positive deflection no matter how small that positive deflection may be is an S wave.



In lead V_1 the classical morphology of the QRS complex is small r wave followed by larger S wave. While in V_6 an initial small negative deflection q wave is followed by large R wave. In the example shown here there is no S wave present in V_6 , also small s wave is seen in this lead in many normally ECGs.

The section of the ECG recording, connecting the end of the QRS complex and the beginning of the T wave is termed the **ST** segment. And the junction between the ST segment and the end of the QRS complex is termed the **J** point.

As all of the ventricular muscle mass is depolarized at this time, there is no current flow through the heart, and the J point and ST segment should therefore lie on the iso-electric line.



This is generally true, but you will learn later that, there are normal ECG variants in which the ST segment lies above the iso-electric line. This becomes very important when we go on to try to identify patients with myocardial infarction.

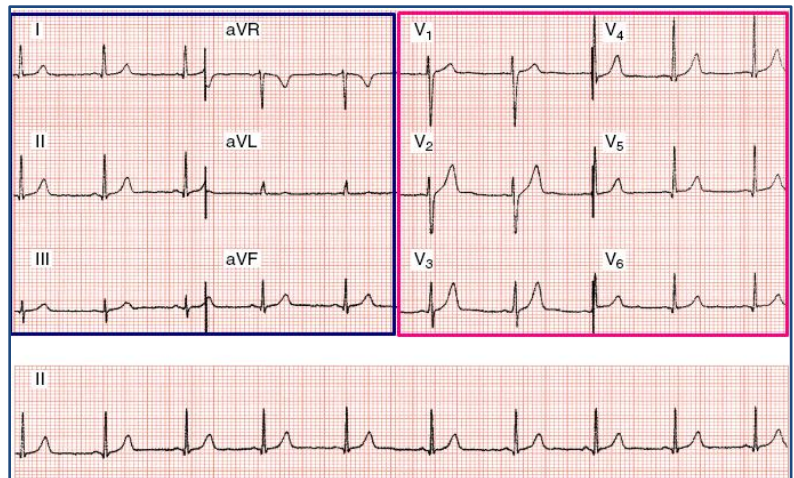
Finally, the diffuse deflection produced by ventricular repolarization is termed the T wave.

ECG Lead Perspectives

Learning the position of the leads of standard ECG relative to the heart is not as difficult as it seems. Consider the 12 leads in two groups of 6.

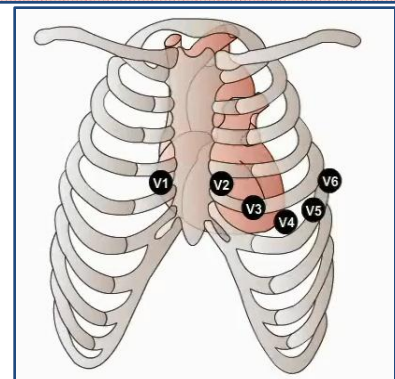
With the 6 chest leads also referred to as the precordial leads examining the heart in horizontal plane.

And second groups of 6 leads which examine the heart in the vertical plane.



We will first deal with the horizontal group (the precordial or chest leads), these 6 leads V₁ to V₆ are placed on the surface of the chest wall.

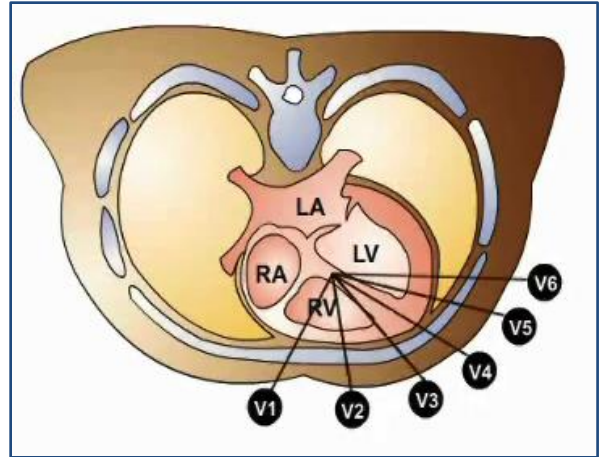
- V₁ in the 4th right intercostals space to the right of the sternum .
- V₂ in the 4th left intercostals space to the left of the sternum.
- V₃ between V₂ and V₄.



- V₄ in the 5th left intercostals space in the mid clavicular line.
- V₅ at the same horizontal level of V₄ but at anterior axillary line in the left.
- V₆ at the same horizontal level of V₄ but at mid axillary line in the left.

These 6 chest leads examine the heart in the horizontal plane. In order to understand the view of cardiac electric activity we need to remind you the position of the heart in the chest cavity.

Let's examine a horizontal section of the chest taking at the level of the chest leads. As illustrated in this section, the heart is positioned such that the right and left atria sit behind the ventricles at the back of the chest. Furthermore the organs is rotated towards the left, so the right ventricle lies anterior to left, immediately behind sternum. The 6 chest leads V₁ to V₆ examine the heart in this horizontal plane. Therefore, V₁ and V₂ face the anterior surface of the right ventricle. V₃ and V₄ look at the anterior surface of the left ventricle. While V₅ and V₆ look at the lateral surface of the left ventricle.



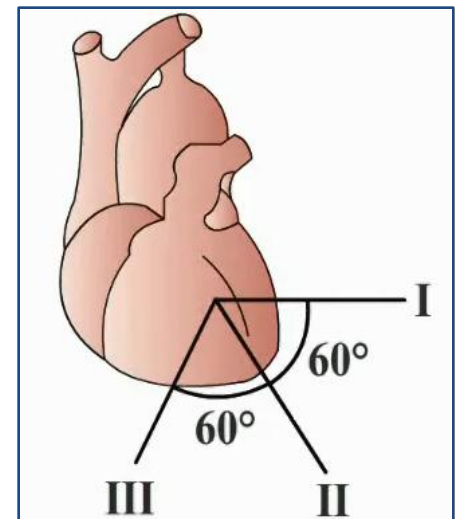
The remaining 6 ECG leads, we can consider in two groups :

- The standard leads (Lead I, II and III).
- The augmented leads (aVR, aVL and aVF).

This vertical plane is known in anatomical terms as the frontal plane. To remember the position of all 6 of the vertical leads, use Lead one as your reference point .

- ✓ Lead one: looks directly at the heart from the patient left hand side and define zero degrees in all for the discussion of the frontal leads.
- ✓ Lead two: looks the heart at an angle 60° clock wise.
- ✓ Lead three: is positioned 60° clock wise from lead two.

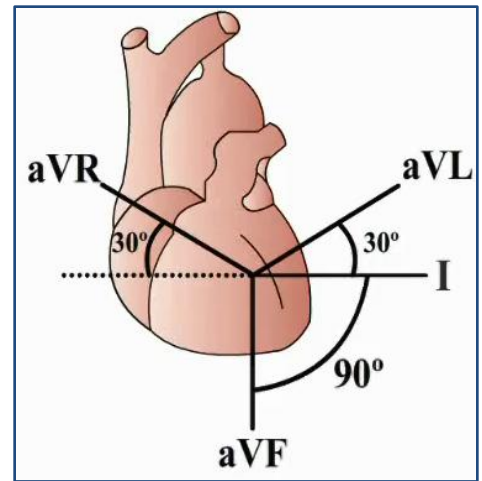
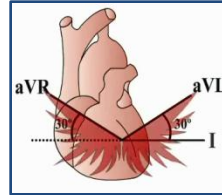
The reader from the standard leads (Lead I, II and III) are recorded on the right hand of the ECG paper.



Now, we will talk about the three further vertical leads (the augmented leads) aVR, aVL and aVF.

- ✓ aVL: looks at the heart from the left 30° anti-clock wise from lead one.
- ✓ aVR: looks at the right side of the heart and just like aVL 30° above the horizontal, relative to lead one.

As aVL and aVR are situated at 30° of the horizontal plane, you can think of them as left and right wings of the ECG



- aVF: looks straight at the inferior surface of the heart and therefore at 90° clock wise from lead one.

N.B.

Now you know the vertical leads two, three and aVF form a group examining the inferior or diaphragmatic surface of the ventricles. A region supplied by right coronary artery.

The chest leads V₁ to V₄ examine the anterior surface of the ventricle and the septum. A region supplied by the left anterior descending artery.

While leads one, aVL, V₅ and V₆ examine the left lateral aspect of the left ventricle. A region supplied by the left circumflex artery.

Topography

The relation between the ECG leads and the walls of the heart

Leads	Wall
II - III - aVF	Inferior
I - aVL	High lateral wall
V ₁ - V ₂	Septal (antro-septal)
V ₃ - V ₄	Strict anterior
V ₅ - V ₆	Low lateral
V ₁ - V ₃ R V ₆ R	RV free wall
Louis Leads	Atrial Activity
N.B. posterior wall potentials are recorded in the anterior leads as a mirror image for waves provided to be drawn in the posterior leads because posterior leads are technically difficult to be made.	

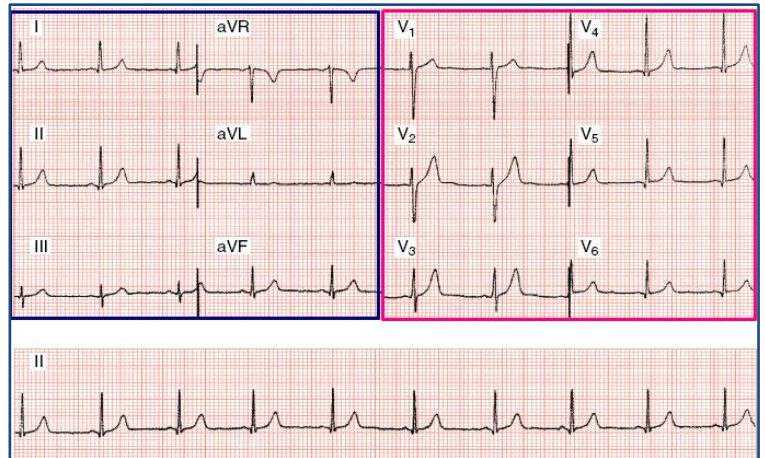
There are some extra chest leads which can be used in cases of dextrocardiaetc.

- V_3R : as V_3 but on right side.
- V_4R : as V_4 but on right side.
- V_5R : as V_5 but on right side.
- V_6R : as V_6 but on right side.

Time and the ECG paper

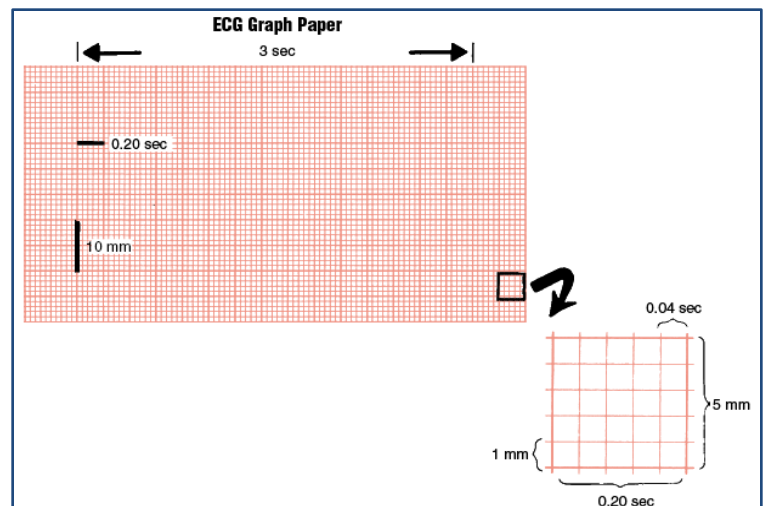
If you look at the bottom of the ECG paper, you will see a long run of recording from lead two, this is called rhythm strip. We use the rhythm strip to calculate the heart rate and to diagnose abnormal cardiac rhythms.

Lead two is used as the rhythm strip as it is usually the easiest lead to show the **P** wave. Which plays a key role in the diagnosis of rhythm disturbances on the ECG.

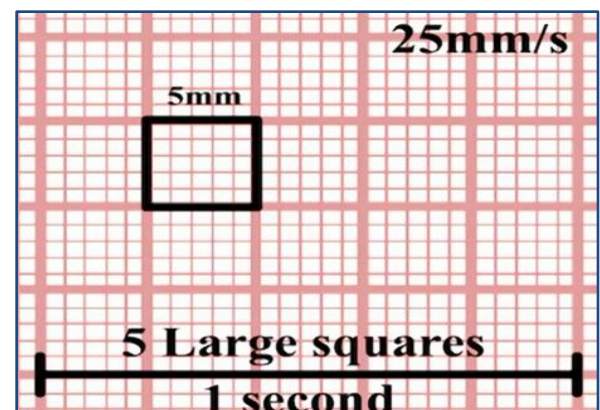


Let's deal with calculation of the Heart rate, ECG recording paper is divided into large squares (5 mm wide), and these large squares are subdivided into small squares (each of 1 mm in width).

We can consider that, the recording needle in the ECG machine, runs at a constant speed over the ECG paper of 25 mm every second.



If you think about it, you will realize that, this means the distance on the ECG paper equates to time. And the recording rate of 25 mm per second >> 5 large squares are covered in one second. So, three hundred large squares represent one minute. Therefore the number of R waves in 300 large squares are the heart rate in beats per minutes .



Look at the rhythm strip on this ECG, there is one R wave present every 5 large squares. So, in 300 large squares there will be 60 R waves present.

This patient's heart rate is therefore 60 beats per minute.



A simple method to calculating heart rate from ECG,

- Identify two R waves on the rhythm strip.
- Count the number of large squares between them (n).
- Divide 300 by this ($300/n$).

Provided that the heart rate is regular, this method gives you an accurate heart rate in beats per minute.

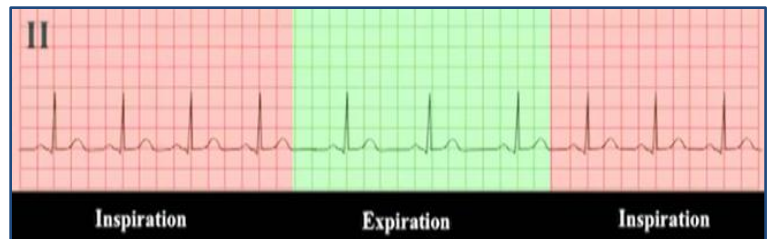
You also noticed that 300 large squares equates one minute, and as there is five small squares in each large square >> 1500 small squares will equate one minute.

Using exactly the same logic as before, therefore we can also calculate the heart rate by counting the number of small squares between consecutive R waves and dividing this number into 1500. This is useful when the R wave does not fall on large square.

You remember from physiology that sinus arrhythmia is completely normal variant, with heart rate slows during expiration and speeds up during inspiration.

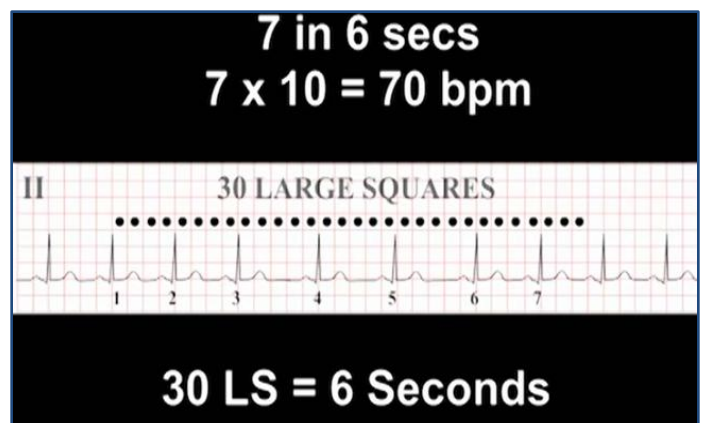
Many normal hearts as illustrated here, the heart rate somewhere irregular due to phenomenon termed sinus arrhythmia.

We cannot use the RR interval technique to calculate heart rate in this case.



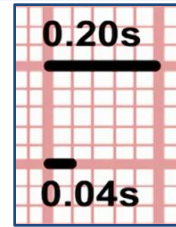
As the RR interval varies. When calculating the heart rate from an ECG in the presence of an irregular rhythm count 30 large squares (remembering that 5 large squares equates to one second - 30 large squares equates to 6 seconds), count the number of R waves in these 30 squares.

In this case they are 7. Seven beats in 6 seconds gives the heart rate of 70 beats per minute.



The standard recording speed of 25 mm per second:

- 5 large squares corresponds to one second.
- One large square corresponds to one fifth of a second.
- One small square corresponds to 0.04 second.



Comment on ECG

We will mention 10 items ☺

1. Rhythm
2. Rate
3. Axis
4. P wave
5. P-R interval
6. QRS complex
7. S-T segment
8. T wave
9. Q-T interval
10. U wave

1. Rhythm

We comment on two things :

- Sinus or not ??
- Regular or irregular ??

What is meant by sinus ??

Every P wave is followed by QRS complex

What is meant by regular ??

Numbers of big squares between each RR interval are equal

What is meant by irregular ??

Numbers of big squares between each RR interval are not equal.

This irregular rhythm may be :

- ✓ Marked irregularity (e.g., atrial fibrillation)
- ✓ Occasional irregularity (e.g., extra systole)

We know the rhythm is regular or irregular from the rhythm strip. If you don't find rhythm strip in the ECG paper, you should search for any lead with more than 3 heart beats and count the numbers of squares between RR interval to see if regular or not.

2. Rate

Normal heart rates ranges from 60 to 90 beats per minute.

- More than 100 beats per minute >> tachyarrhythmia
- Less than 60 beats per minute >> bradyarrhythmia

How to calculate the heart rate ??

First look at the rhythm :

- If regular rhythm >> the heart rate equates $300 / n$ (which n the number of big squares between RR interval).
Or $1500 / n$ (which n the number of small squares between RR interval), more accurate.
- If irregular rhythm >> count the number of R waves in 30 big squares and multiply the result by 10.
Or, $300 / n$ (which n the average number of RR interval)

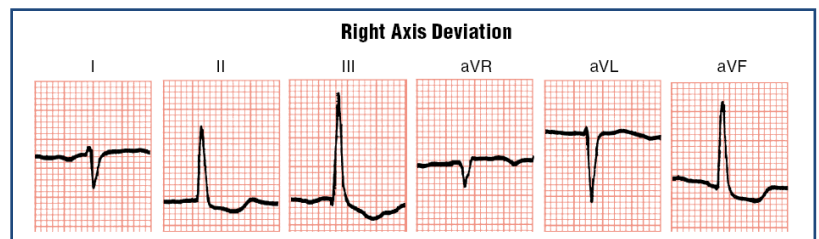
You must comment on the rhythm before the rate ☺

3. Axis

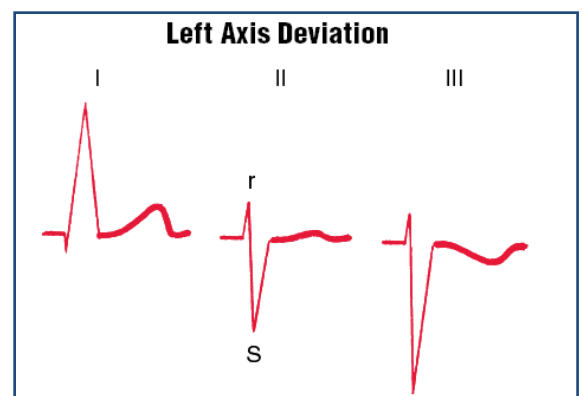
Look at QRS complex in lead one and aVF (or lead two).
Normally QRS complex is positive in lead one and aVF .



If you found the QRS complex is negative in lead one and positive in aVF this means right axis deviation.



If you found the QRS complex is positive in lead one and negative in aVF (lead II) this means left axis deviation.

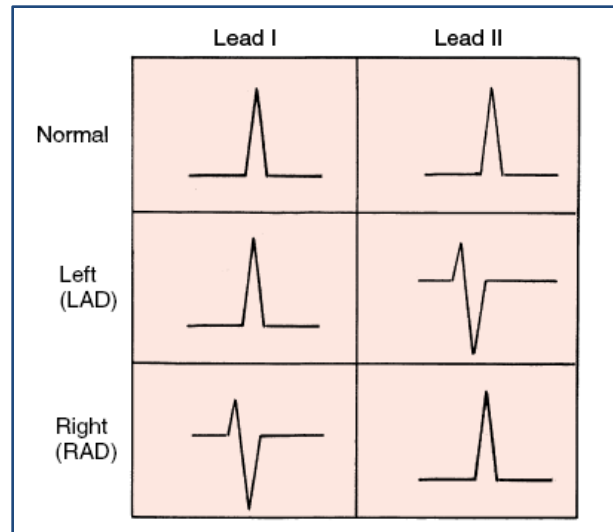


Take care ☺ in oral exam,

Types of axis deviation ??

- Right axis deviation.
- Left axis deviation.

Normal axis is not deviated ☺



Causes of right axis deviation

- Children
- Tall thin adults
- Right ventricular hypertrophy
- Chronic lung disease
- Anterolateral myocardial infarction
- Pulmonary embolus
- Atrial septal defect
- Ventricular septal defect

Causes of left axis deviation

- Q waves of inferior MI
- Artificial cardiac pacing
- Left ventricular hypertrophy
- Hyperkalemia
- Ostium primum ASD
- Injection of contrast into left coronary artery

Note : pt. of left ventricular hypertrophy not usually has LAD

4. P wave

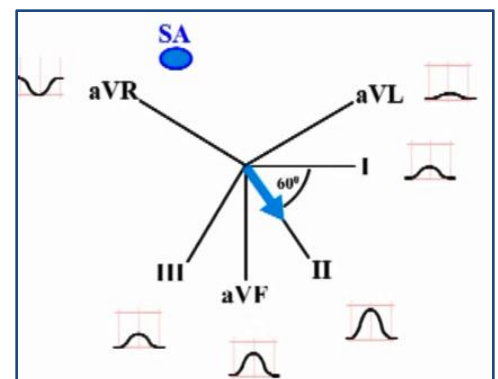
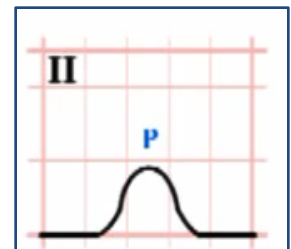
The P wave represent the atrial depolarization. It is the first positive wave before the complex.

There some features in the process of spread of depolarization through the atrial chamber which we would like you to know.

Atrial depolarization moves through the chambers downwards and towards the left from the SA node. The normal P wave axis is indicated here by the blue arrow. (i.e., downwards and leftwards) travels more or less straight down to lead II in the frontal plane. Hence you can see here P wave originating from a sinus discharge are usually strongly positive in the inferior leads having maximum amplitude in lead II.

Also with an axis $+60^\circ$ the P wave is positive in most of frontal leads. And of course negative in the aVR

So, P wave is better to seen in Lead II and V_1 .



- ✚ **Width (duration) :** = < 2.5 small square (< 0.12 sec.).
- ✚ **Height (amplitude) :** = < 2.5 small square (< 2.5 mm).

The P wave has two possibilities :

- Present
- Absent

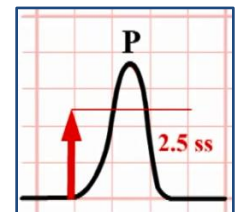
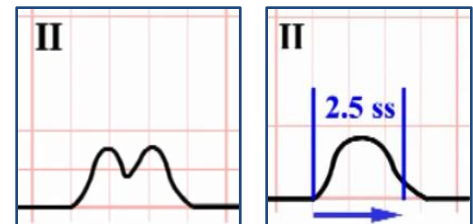
If the P wave is present, it has two possibilities :

- Normal (less than 2.5 X 2.5 small squares)
- Abnormal

00

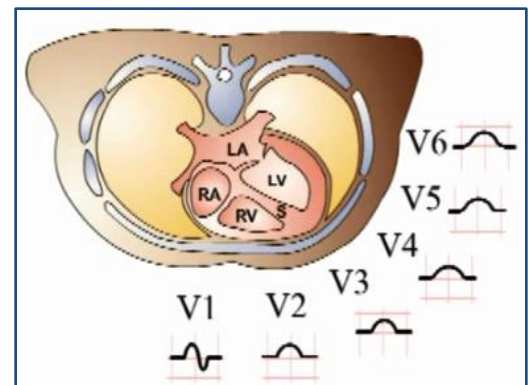
What is the possibilities of abnormal P wave ??

1. Broad P wave (M shaped- P mitrale)
where the P wave becomes broad (> 2.5 small squares)
denotes left atrial strain.
2. Peaked and high voltage P (P pulmonale)
where the P wave becomes tall and peaked (> 2.5 small squares)
denotes right atrial strain.
3. Pulmonale Mitral
where the P wave is tall and broad (> 2.5 X 2.5 small squares)
4. Biphasic
where part of the P wave is positive and the other is negative

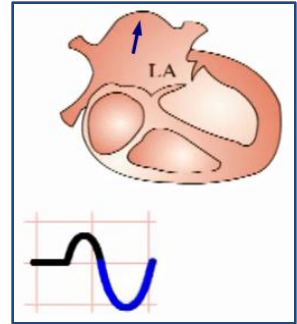


The P wave in V₁ is biphasic (the first part represent the right atrium and second part represent the left atrium)

The spread of the atrial depolarization is less prominent in the horizontal plane compared to frontal plane. As the left atrium lies somewhat posterior to the right chamber, left atrial depolarization moving posteriorly and to the left may produce small negative terminal deflection in the P wave recorded in lead V₁ this is observed in many normal ECG.



However, in the presence of left atrial enlargement this finding can be dramatically exaggerated. Enlargement in the chamber is usually directed posteriorly and to the left and this can result in very prominent negative terminal component to the P wave in lead V_1 .



If the P wave is absent

Look at the rhythm ☺ :

- Irregular (A.F.)
- Regular

Absent P wave with regular rhythm look at QRS complex

- ✓ Wide QRS complex (> 3 small squares) :

- ✚ Ventricular tachycardia
- ✚ Ventricular fibrillation

- ✓ Narrow QRS complex :

- ✚ Supra ventricular tachycardia
- ✚ Nodal rhythm

How to differentiate between them ??

By rate :

- ✚ Supra ventricular tachycardia >> tachycardia.
- ✚ Nodal rhythm >> slow

Don't forget,

If you see sawtooth appearance >> Atrial flutter

5. P-R interval

The normal heart, the time between the onset of atrial depolarization (the beginning of P wave) and the onset of ventricular depolarization (the beginning of the QRS complex) varies between 0.12 second to 0.2 second (between 3 and 5 small squares) this is PR interval

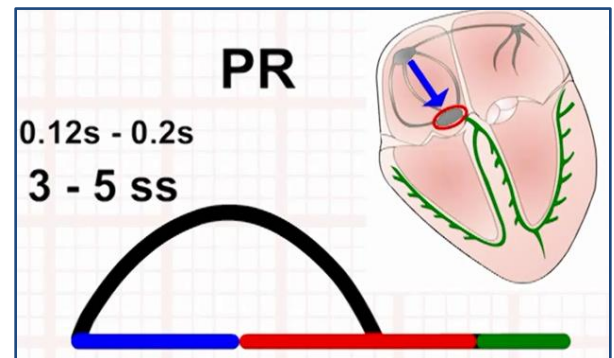


The PR interval is made up of a number of elements :

- The first component represented here in (blue) is the time taken for the depolarization wave (normally generated from the SA node to the atria and reach the AV node)

You know the depolarization reach the AV node before the end of the P wave. However, the AV node delays the transit of impulses to the ventricles.

- This physiological delay in the AV node is the second major component in the PR interval. (red)
- The third part contributing in the PR interval shown here in (green) is the time taking by the depolarization wave to transit through the bundle of His and the branches of the interventricular conducting system.



Better to seen in lead two.

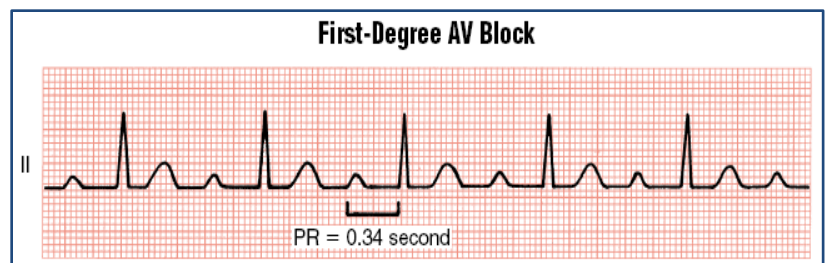
What is the possibilities of PR interval ??

- Normal (between 3 and 5 small squares)
- Prolonged (> 5 small squares)
- Shortened (< 3 small squares)

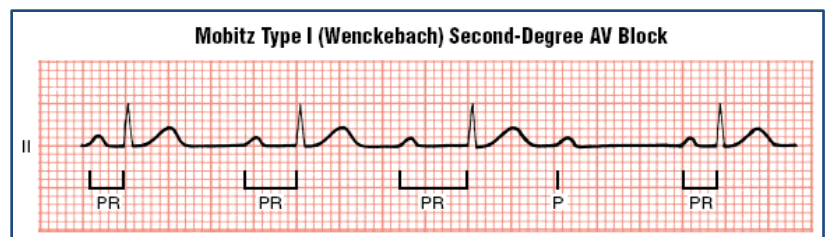
Prolonged PR interval

1. PR interval (long and fixed)
just prolonged PR interval

First degree heart block



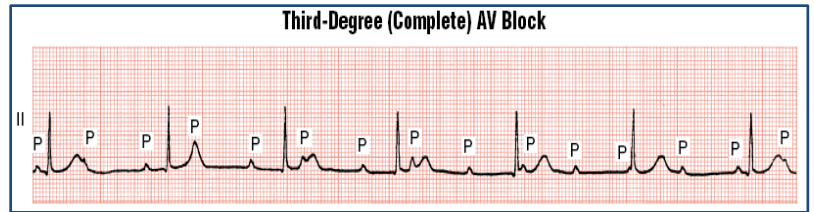
2. PR interval (long with fixed change)
progressive prolongation of PR interval until dropped beat
Wenckebach phenomena
urgent case ☹



3. PR interval (not fixed)(variable)
there is no relation between the atria and ventricle
Atrio-ventricular dissociation

Complete heart block

here,
if the P wave is present before the complex, it happens by chance.



Shortened PR interval

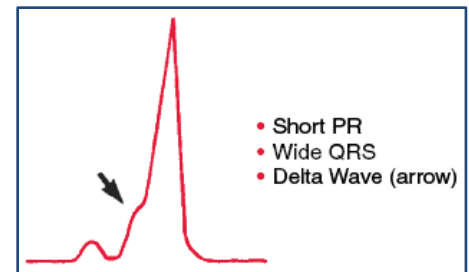
Wolff-Parkinson-White

Normally the electrical stimulus travels to the ventricles from the atria via the atrioventricular (AV) junction. The physiologic lag of conduction through the AV junction results in the normal PR interval of 0.12 to 0.2 sec. Consider the consequences of having an extra pathway between the atria and ventricles that would bypass the AV junction and preexcite the ventricles. This situation is exactly what occurs with the WPW pattern: an atrioventricular bypass tract connects the atria and ventricles, circumventing the AV junction. Bypass tracts (also called accessory pathways) represent persistent abnormal connections that form and fail to disappear during fetal development of the heart in certain individuals.

These abnormal conduction pathways, composed of bands of heart muscle tissue, are located in the area around the mitral or tricuspid valves (AV rings) or interventricular septum. An AV bypass tract is sometimes referred to as a bundle of Kent.

Preexcitation of the ventricles with the classic WPW pattern produces the following characteristic triad of findings on the ECG :

1. Short P-R interval
2. Wide QRS complex
3. Delta wave



There are two types of Wolff-Parkinson-White:

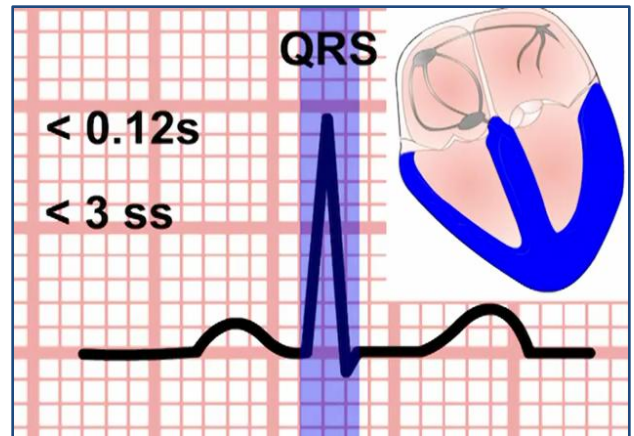
- Type A >> left ventricular pattern (more dangerous)
- Type B >> right ventricular pattern

6. QRS complex

The QRS complex represents the spread of a stimulus through the ventricles.

Better to be seen in :

- Right ventricle ($V_{1,2}$)
- Left ventricle ($V_{5,6}$)
- ✚ Q wave >> first negative wave in the complex
- ✚ R wave >> first positive wave in the complex
- ✚ S wave >> the negative wave following R



Q wave

is the first negative wave in the complex

- ✚ Width : less than one small square
- ✚ Height : less than $\frac{1}{4}$ the following R wave

N.B. Pathological Q :

Where the Q wave is deep and wide (does not seen in normal ECG) (present in Myocardial infarction)

If you found pathological Q, you should search for topographism :

- If in $V_{1,2}$ >> anterior infarction
- If in $V_{3,4}$ >> septal infarction
- If in $V_{5,6}$ >> Lateral infarction
- If in $V_{1,2,3,4}$ >> antro-septal infarction
- If in $V_{1,2,3,4,5}$ >> Extensive anterior infarction

Pathological Q can be found normally in ECG ???

Yes, in aVR as it draws the cavity of the heart and aVL in case of dextrocardia

Sometimes, in V_1 r wave is small to the extent that you feel it is absent and confuse with S and Q wave >> So, don't comment on pathological Q in V_1 and aVR.

In Myocardial infarction >> anterior infarction in V_1 and V_2 (not only V_1)

R wave

The first positive wave in the complex (you may say the only positive wave in the complex)

Used as voltage criteria

- ✚ Width : between two and three small squares
- ✚ Height : between one and five big squares

Wide R wave (> 3 small squares “wide complex” in cases of LBBB, RBBB, Ventricular tachycardia)

S wave

It is the first negative wave following R

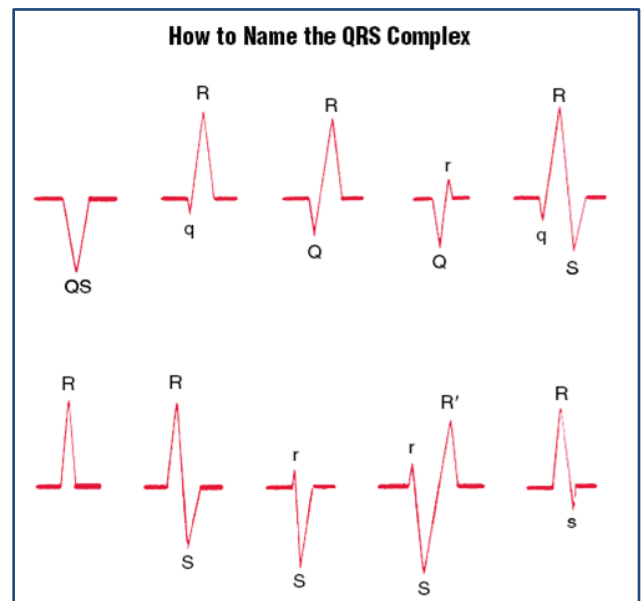
There is a relation between S and R waves in chest leads :

- S wave starts big in V_1 and gradually decreases till V_5
- On the contrary R wave starts small in V_1 and gradually increases till V_6

N.B.

- **S** in V_2 is > **S** in V_1
- **S** progress from V_2 to V_5
- **S** usually absent in V_6

One of the most confusing aspects of electrocardiography for the beginning student is the nomenclature of the QRS complex. However, not every QRS complex contains a Q wave, an R wave, and an S wave hence the confusion. The bothersome but unavoidable nomenclature becomes understandable if you remember several basic features of the QRS complex. When the initial deflection of the QRS complex is negative (below the baseline), it is called a Q wave. The first positive deflection in the QRS complex is called an R wave. A negative deflection following the R wave is called an S wave.



N.B.

- ❖ If the amplitude of the wave less than 5 mm (< 5 small squares) >> written in small letter.
- ❖ If the amplitude of the wave more than 5 mm (> 5 small squares) >> written in capital letter.

Not every “QRS” contain “Q”, “R” & “S”, but it may be :

- Monophasic (R or QS)
- Biphasic (RS or QR)
- Triphasic (QRS or RSR')

< 1 big square (low voltage)

- Terminal heart failure
- Cardiomyopathy
- IHD
- Obesity
- Emphysema
- Pericardial effusion

> 5 big squares (high voltage)

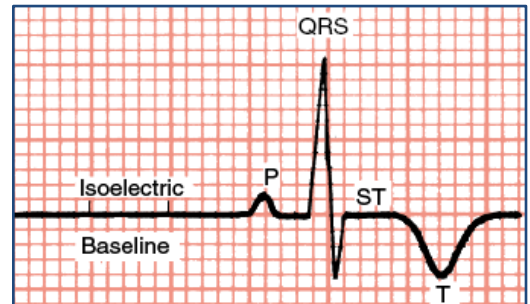
- Ventricular hypertrophy

7. S-T segment

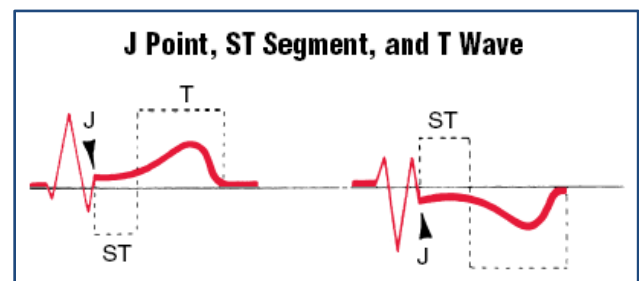
The ST segment is that portion of the ECG cycle from the end of the QRS complex to the beginning of the T wave.

It represents the beginning of ventricular repolarization. The normal ST segment is usually isoelectric (i.e., flat on the baseline, neither positive nor negative), but it may be slightly elevated or depressed normally (usually by less than 1 mm).

Some pathologic conditions such as myocardial infarction (MI) produce characteristic abnormal deviations of the ST segment. The very beginning of the ST segment (actually the junction between the end of the QRS complex and the beginning of the ST segment) is sometimes called the J point.

**J point**

- Point where QRS complex returns to iso-electric line.
- Beginning of S-T segment.
- Critical in measuring S-T elevation.



You should check ST segment in all leads.

What is the possibilities of ST segment ??

- Iso-electric line
- Elevated
- Depressed

Iso-electric line

is the base line on an electrocardiogram (PR or TP line)

S-T elevation

What is the causes of the ST elevation (above the iso-electric line) ??

- Pericarditis
- Myocardial infarction
- Prinzmetal's angina

How to differentiate between them ??

- ✓ In precarditis ST segment elevation >> in all leads
- ✓ Angina & Myocardial infarction >> in some leads

How to differentiate between angina and myocardial infarction ??

- ✓ Cardiac enzymes >> elevated in myocardial infarction
- ✓ Timing >> ST elevation more than half an hour >> myocardial infarction

S-T depression

What is the causes of ST depression ??

- Digitalis
- Hypokalemia
- Angina (better to say ischemia as angina is a clinical diagnosis)
- Myocardial infarction
- Pericarditis
- Cardiac hypertrophy
- Bundle branch block

How to differentiate between them ??

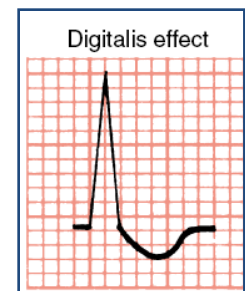
- Digitalis
- Hypokalemia
- Pericarditis

With diffuse ST segment depression in all leads

Digitalis : the ST segment depression with J point at iso-electric line (called sagging)

Hypokalemia : measuring the serum potassium

Pericarditis : clinically by stitch pain



If ST segment depression in some leads :

- Angina (better to say ischemia as angina is a clinical diagnosis)
- Myocardial infarction
- Cardiac hypertrophy
- Bundle branch block

As Known V_1, V_2 and V_3 >> the leads of the right ventricle,

So ,

the ST segment depression in V_1, V_2 and V_3 >> with right ventricular hypertrophy >> strain pattern (or secondary changes)

Left ventricular enlargement >>

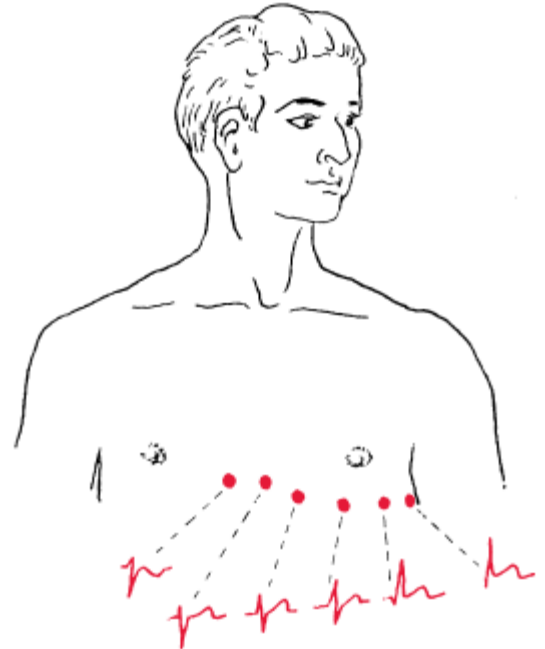
ST segment depression in V_4, V_5 and V_6

Right bundle branch block >>

ST segment depression in V_1, V_2 and V_3

Left bundle branch block >>

ST segment depression in V_4, V_5 and V_6



Don't forget ☺

- ❖ If you found rSR' in V_1 (right bundle branch block) check for ST segment in V_1, V_2 and V_3 >> if depressed >> right bundle branch block or right ventricular hypertrophy
- ❖ If there is left ventricular enlargement >> check for ST segment in V_4, V_5 and V_6 >> If depressed >> secondary changes due to left ventricular hypertrophy
- ❖ If you don't find ventricular hypertrophy or bundle branch block >> Angina (better to say ischemia as angina is a clinical diagnosis)

The J point in ischemia is below the iso-electric line (while in digitalis >> J point is iso-electric line)

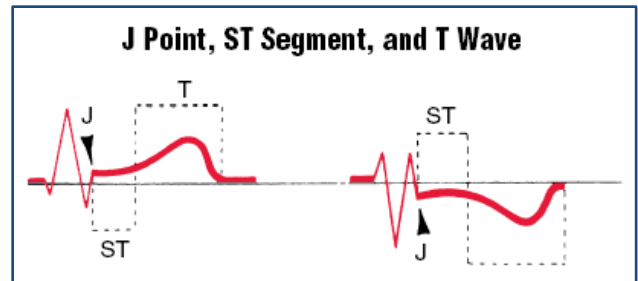
How the precarditis causes ECG changes, as we know ECG record cardiac muscle changes ???!
Precarditis always associated with very superficial myocarditis

8. T wave (Never absent)

The T wave represents part of ventricular repolarization. A normal T wave has an asymmetrical shape; that is, its peak is closer to the end of the wave than to the beginning.

When the T wave is positive, it normally rises slowly and then abruptly returns to the baseline. When it is negative, it descends slowly and abruptly rises to the baseline.

The asymmetry of the normal T wave contrasts with the symmetry of T waves in certain abnormal conditions, such as MI and a high serum potassium level.



- ✚ Width : less than 6 small squares
- ✚ Height : less than 1/3 the preceding R wave

What is the possibilities ??

- Upright (positive)
- Inverted (negative)

Positive T wave

- ✓ Normal
- ✓ Hyperacute >> called Himalaya T in cases of hyperkalemia

Inverted T wave

- ✓ May be normal in some individuals

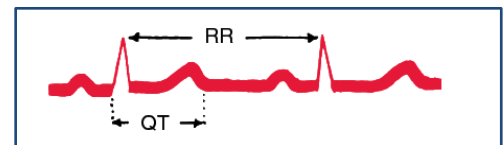
T wave inversion is insignificant per se, its significance appears if it became upright in which case would be called dynamic T which is dangerous sign

9. Q-T interval

The QT interval is measured from the beginning of the QRS complex to the end of the T wave.

It primarily represents the return of stimulated ventricles to their resting state (ventricular repolarization).

The normal values for the QT interval depend on the heart rate. As the heart rate increases (RR interval shortens), the QT interval normally shortens; as the heart rate decreases (RR interval lengthens), the QT interval lengthens. The QT should be measured in the ECG lead that shows the longest intervals. A common mistake is to limit this measurement to lead II. You can measure several intervals and use the average value. When the QT interval is long, it is often difficult to measure because the end of the T wave may merge imperceptibly with the U wave. As a result, you may be measuring the QU interval, rather than the QT interval



Normally >> 11 small squares (0.44 seconds)

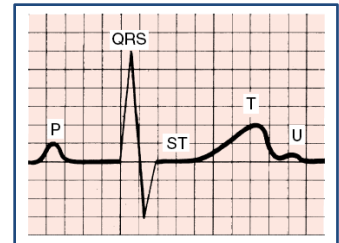
Long Q-T interval

- Drugs (many antiarrhythmics, tricyclics & phenothiazines)
- Electrolyte abnormalities (K^+ , Ca^{++} , Mg^{++})
- CNS disease (especially subarachnoid hemorrhage, stroke, trauma)
- Hereditary LQT

10. U wave

The U wave is a small, rounded deflection sometimes seen after the T wave. Its exact significance is not known.

Functionally, U waves represent the last phase of ventricular repolarization. Prominent U waves are characteristic of hypokalemia. Very prominent U waves may also be seen in other settings, for example, in patients taking drugs such as sotalol or one of the phenothiazines or sometimes after patients have had a cerebrovascular accident.



The appearance of very prominent U waves in such settings, with or without actual QT prolongation, may also predispose patients to ventricular arrhythmias. Normally the direction of the U wave is the same as that of the T wave. Negative U waves sometimes appear with positive T waves. This abnormal finding has been noted in left ventricular hypertrophy and myocardial ischemia.

Abnormal ECG

We will mention five items ☺

1. Chamber enlargement
2. Bundle branch block (BBB)
3. Coronary ischemia (MI & ischemia)
4. Heart block
5. Others

1. Chamber enlargement

Divided into:

- Atrial enlargement
- Ventricular enlargement

The atrial enlargement is further divided into :

- ✓ Right atrial enlargement
- ✓ Left atrial enlargement

The ventricular enlargement is further divided into :

- ✓ Right ventricular enlargement
- ✓ Left ventricular enlargement

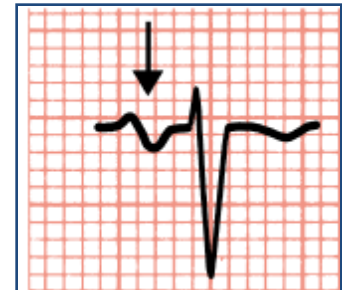
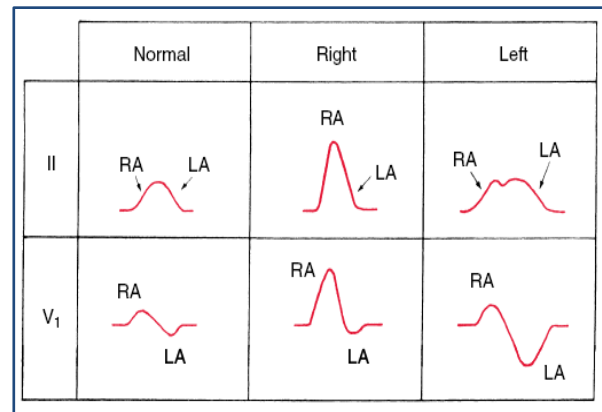
Atrial enlargement

✚ P wave >> tall and peaked (> 2.5 small squares) (called P pulmonal) >> right atrial enlargement

✚ P wave >> broad (> 2.5 small squares) >> left atrial enlargement

✚ P wave >> biphasic in V_1

- ✓ first part represent the right atrium (positive part) >> if enlarged >> right atrial enlargement
- ✓ second part represent the left atrium (negative part) >> if enlarged >> left atrial enlargement



Ventricular enlargement

Check QRS in $V_{1,2,5,6}$

- ✚ Normally in $V_{1,2}$ >> S wave bigger than r wave
- ✚ Normally in $V_{5,6}$ >> R wave bigger than s wave

If you find in $V_{1,2}$ S wave bigger than r wave, but the S wave is so deep (exaggeration of normal) (S wave more than 5 big squares) >> left ventricular hypertrophy

Voltage criteria of exaggeration of normal :

- ❖ S wave > 5 big squares in V_1 or V_2
- ❖ R wave > 5 big squares in V_5 or V_6
- ❖ The summation of S + R waves ≥ 7 big squares

This is means left ventricular enlargement

As the cardiac muscle hypertrophied and the blood supply didn't change so, the cardiac muscle will show some changes (strain ischemia) :

- Depressed ST segment
- Inverted T wave
- Or one of them

These changes will take place in lead V_5 and V_6 as we are talking about left ventricle

How to know that the right ventricle is enlarged ?

If you found reversal of normal (R wave > s wave in $V_{1,2}$) or (S wave > r wave in $V_{5,6}$)

N.B.

I can diagnose right ventricle enlargement from one lead only (V_1, V_2, V_5, V_6)

As the cardiac muscle hypertrophied and the blood supply didn't change so, the cardiac muscle will show some changes (strain ischemia) :

- Depressed ST segment
- Inverted T wave
- Or one of them

These changes will take place in lead V_1 and V_2 as we are talking about right ventricle

If someone has biventricular hypertrophy,

The strain ischemia will appear in V_1, V_2 and appears also in V_5 and V_6

2. Bundle Branch Block (BBB)

We will talk about :

- Right bundle branch block
- Left bundle branch block

How to detect bundle branch block ?

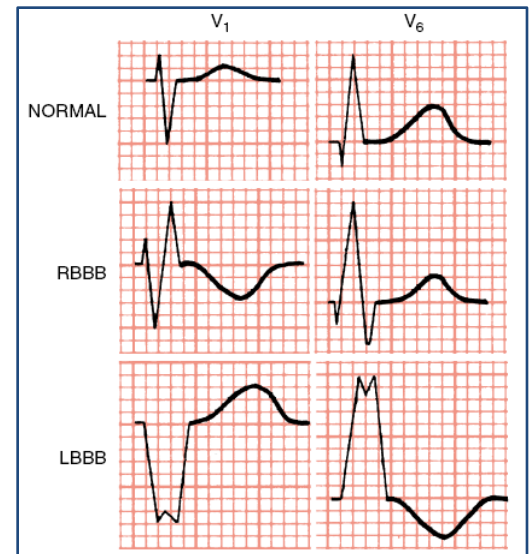
Look at QRS complex, you will find the shape of M (RSR')

- ✚ M shaped in V_1 or V_2 >> right bundle branch block
- ✚ M shaped in V_5 or V_6 >> left bundle branch block

QRS complex here is wide (> 3 small squares)

What are the benefits of QRS complex ?

- Shape
- Direction
- Voltage



I mean, look at the QRS complex checking the shape, direction and the voltage

Shape :

- M shaped >> bundle branch block

if normal shaped, look at direction

Direction :

- Reversal of normal >> right ventricular enlargement

If normal shaped and normal direction, look at the voltage

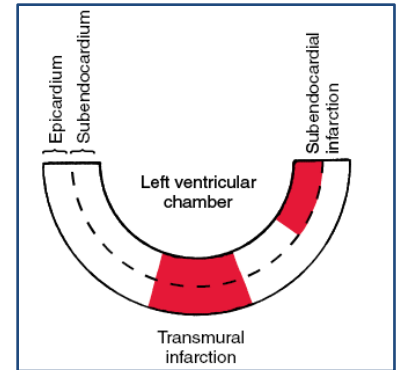
Voltage :

Exaggeration of normal >> left ventricular enlargement

3. Coronary Ischemia (MI & ischemia)

Myocardial cells require oxygen and other nutrients to function. Oxygenated blood is supplied by the coronary arteries. If severe narrowing or complete blockage of a coronary artery causes the blood flow to become inadequate, ischemia of the heart muscle develops. Ischemia means literally “to hold back blood.” Myocardial ischemia may occur transiently. For example, patients who experience angina pectoris with exercise are having transient myocardial ischemia. If the ischemia is more severe, necrosis of a portion of heart muscle may occur. Myocardial infarction (MI) refers to myocardial necrosis (or, in nontechnical parlance—a “heart attack”).

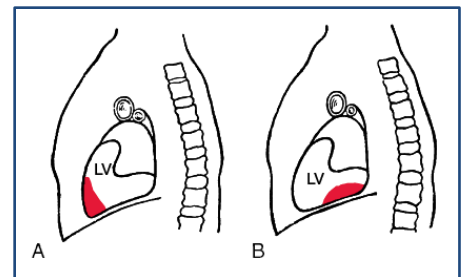
The ventricle consists of an outer layer (epicardium or subepicardium) and an inner layer (subendocardium). This distinction is important because myocardial ischemia may be limited to just the inner layer, or it may affect virtually the entire thickness of the ventricular wall (transmural ischemia).



“Transmural” MI is characterized by ischemia and ultimately necrosis of a portion of the entire (or nearly the entire) thickness of the ventricular wall. Most patients who present with acute MI have underlying atherosclerotic coronary artery disease. The pathophysiology of acute STEMI and subsequent evolving Q wave MI most often relates to occlusion of one of the coronary arteries by a ruptured atherosclerotic plaque, followed by the formation of a clot at this site. The clot is composed of platelets and fibrin, blocking the blood flow downstream. Other factors can cause or contribute to acute STEMI, including cocaine, coronary artery dissections (spontaneous or induced during interventional procedures), coronary emboli, and other factors. Not surprisingly, large transmural MIs generally produce changes in both myocardial depolarization (QRS complex) and myocardial repolarization (ST-T complex).

The earliest ECG changes seen with an acute transmural ischemia/infarction typically occur in the ST-T complex in sequential phases:

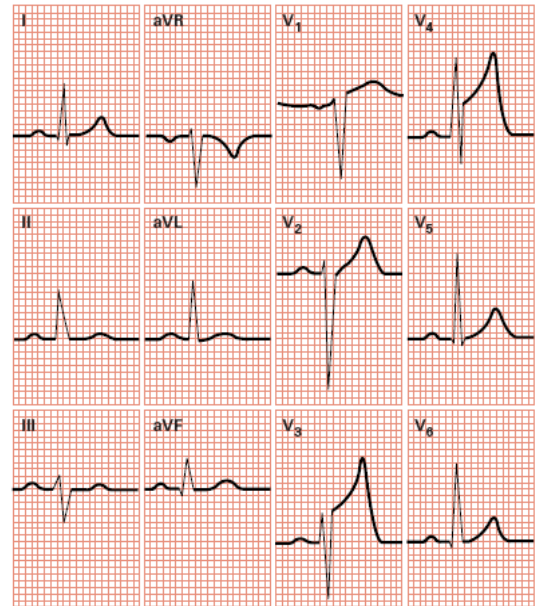
1. The acute phase is marked by the appearance of ST segment elevations and sometimes tall positive (hyperacute) T waves in multiple (usually two or more) leads. The term “STEMI” refers to this phase.
2. The evolving phase occurs hours or days later and is characterized by deep T wave inversions in the leads that previously showed ST elevations. Transmural MIs can also be described in terms of the location of the infarct. Anterior means that the infarct involves the anterior or lateral wall of the left ventricle, whereas inferior indicates involvement of the inferior (diaphragmatic) wall of the left ventricle.



The anatomic location of the infarct determines the leads in which the typical ECG patterns appear. For example, with an acute anterior wall MI, the ST segment elevations and tall hyperacute T waves appear in one or more of the anterior leads (chest leads V₁ to V₆ and extremity leads I and aVL).

Anterior Myocardial Infarction

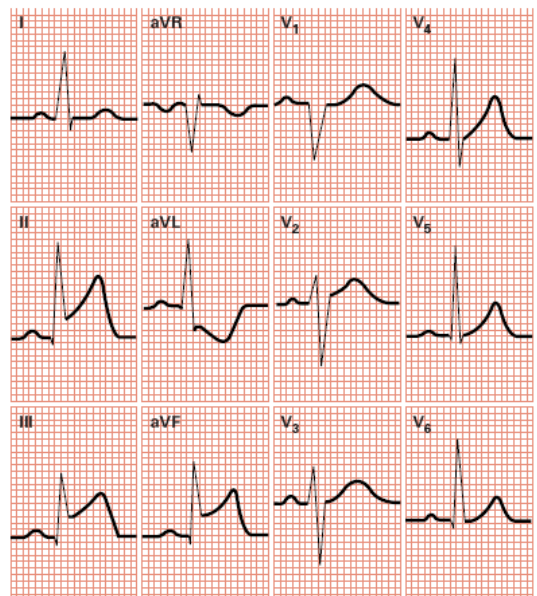
- Occlusion of the left coronary artery—left anterior descending branch
- ECG changes: ST segment elevation with tall T waves and taller-than-normal R waves in leads V₃ and V₄; reciprocal changes in II, III, and aVF



With an inferior wall MI the ST segment elevations and tall hyperacute T waves are seen in inferior leads II, III, and aVF.

Inferior Myocardial Infarction

- Occlusion of the right coronary artery—posterior descending branch
- ECG changes: ST segment elevation in leads II, III, and aVF; reciprocal ST segment depression in I and aVL



An important (but not always present) feature of the ST-T changes seen with STEMI is their reciprocity. The anterior and inferior leads tend to show inverse patterns. Thus in an anterior infarction with ST segment elevations in two or more of leads V₁ to V₆, I, and aVL, ST segment depression is often seen in leads II, III, and aVF.

Conversely, with an acute inferior wall infarction, leads II, III, and aVF show ST segment elevation, with reciprocal ST depressions often seen in one or more of leads V₁ to V₃, I, and aVL.

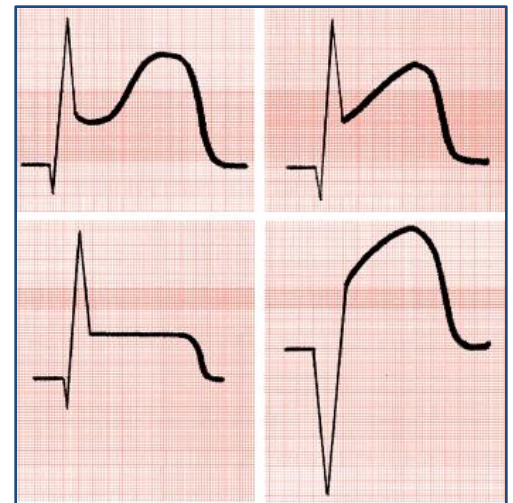
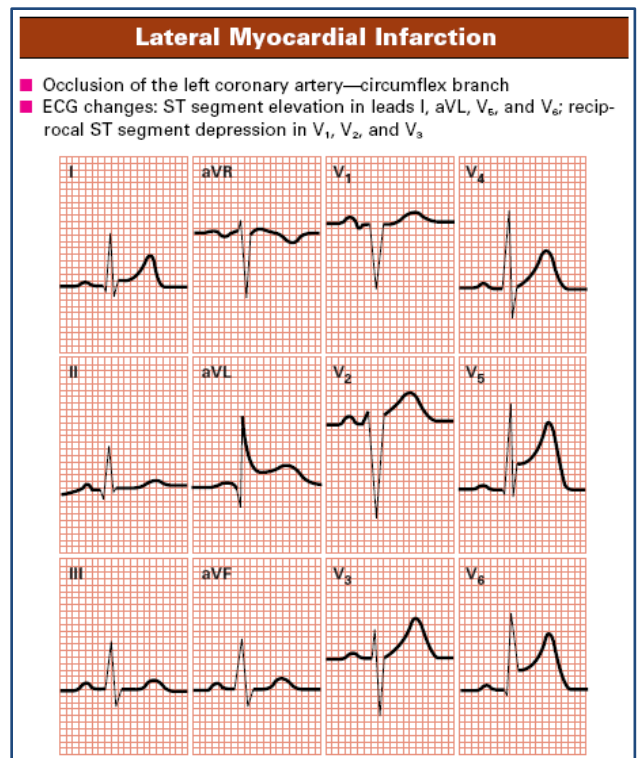
The ST segment elevation seen with acute MI is called a current of injury and indicates that damage has occurred to the epicardial (outer) layer of the heart as a result of severe ischemia. The exact reasons that acute MI produces ST segment elevation are complex and not fully understood.

Normally the ST segment is isoelectric (neither positive nor negative) because no net current flow is occurring at this time. MI alters the electrical charge on the myocardial cell membranes in a number of ways. As a result, current flow becomes abnormal (current of injury) and produces ST segment deviations.

The ST segment elevation seen with acute MI may have different shapes and appearances. It may be plateau-shaped or dome-shaped. Sometimes it is obliquely elevated.

The ST segment elevations (and reciprocal ST depressions) are the earliest ECG signs of infarction, and are generally seen within minutes of blood flow occlusion. Tall, positive (hyperacute) T waves may also be seen at this time. These T waves have the same significance as the ST elevations. In some cases, hyperacute T waves actually precede the ST elevations.

Clinicians should be aware that ST changes in acute ischemia may evolve with the patient under observation. If the initial ECG is not diagnostic of STEMI but the patient continues to have symptoms consistent with myocardial ischemia, serial ECGs at 5- to 10-minute intervals (or continuous 12-lead ST segment monitoring) should be performed. After a variable time lag (usually hours to a few days) the elevated ST segments start to return to the baseline. At the



same time the T waves become inverted in leads that previously showed ST segment elevations.

This phase of T wave inversions is called the evolving phase of the infarction. Thus with an anterior wall infarction the T waves become inverted in one or more of the anterior leads (V_1 to V_6 , I, aVL). With an inferior wall infarction the T waves become inverted in one or more of the inferior leads (II, III, aVF).

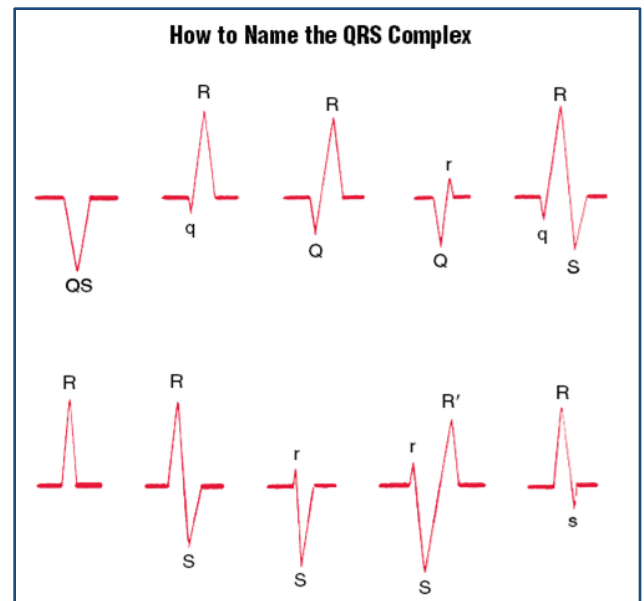
MI, particularly when large and transmural, often produces distinctive changes in the QRS (depolarization) complex.

The characteristic depolarization sign is the appearance of new Q waves.

Why do certain MIs lead to Q waves?

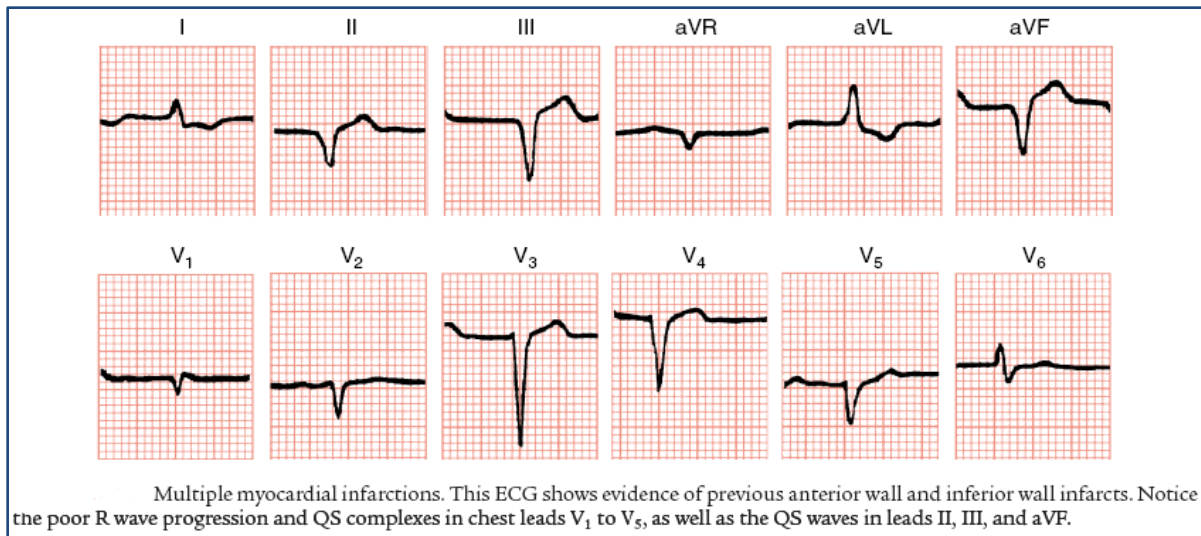
Recall that a Q wave is simply an initial negative deflection of the QRS complex. If the entire QRS complex is negative, it is called a QS complex:

A Q wave (negative initial QRS deflection) in any lead indicates that the electrical voltages are directed away from that particular lead. With a transmural infarction, necrosis of heart muscle occurs in a localized area of the ventricle. As a result the electrical voltages produced by this portion of the myocardium disappear. Instead of positive (R) waves over the infarcted area, Q waves are often recorded (either a QR or QS complex). The common clinical tendency to equate pathologic Q waves with transmural necrosis is an oversimplification. Not all transmural infarcts lead to Q waves, and not all Q wave infarcts correlate with transmural necrosis.



In summary, abnormal Q waves are characteristic markers of infarction. They signify the loss of positive electrical voltages caused by the death of heart muscle. The new Q waves of an MI generally appear within the first day or so of the infarct. With an anterior wall infarction these Q waves are seen in one or more of leads V_1 to V_6 , I, and aVL. With an inferior wall MI the new Q waves appear in leads II, III, and aVF.

Not infrequently, patients may have two or more MIs at different times. For example, a new anterior wall infarct may develop in a patient with a previous inferior wall infarction. In such cases the ECG initially shows abnormal Q waves in leads II, III, and aVF. During the anterior infarct, new Q waves and ST-T changes appear in the anterior leads.



The diagnosis of infarction is more difficult when the patient's baseline ECG shows a bundle branch block pattern or a bundle branch block develops as a complication of the MI. Then the ECG picture becomes more complex.

Remember that RBBB affects primarily the terminal phase of ventricular depolarization, producing a wide R' wave in the right chest leads and a wide S wave in the left chest leads. MI affects the initial phase of ventricular depolarization, producing abnormal Q waves. When RBBB and an infarct occur together, a combination of these patterns is seen: The QRS complex is abnormally wide (0.12 sec or more) as a result of the bundle branch block, lead V₁ shows a terminal positive deflection, and lead V₆ shows a wide S wave. If the infarction is anterior, the ECG shows a loss of R wave progression with abnormal Q waves in the anterior leads and characteristic ST-T changes. If the infarction is inferior, pathologic Q waves and ST-T changes are seen in leads II, III, and aVF.

The diagnosis of LBBB in the presence of MI is considerably more complicated and confusing than that of RBBB. The reason is that LBBB interrupts both the early and the late phases of ventricular stimulation. It also produces secondary ST-T changes.

As a general rule, LBBB hides the diagnosis of an infarct. Thus a patient with a chronic LBBB pattern who develops an acute MI may not show the characteristic changes of infarction. Occasionally, patients with LBBB manifest primary ST-T changes indicative of ischemia or actual infarction. The secondary T wave inversions of uncomplicated LBBB are seen in leads V₄ to V₆ (with prominent R waves).

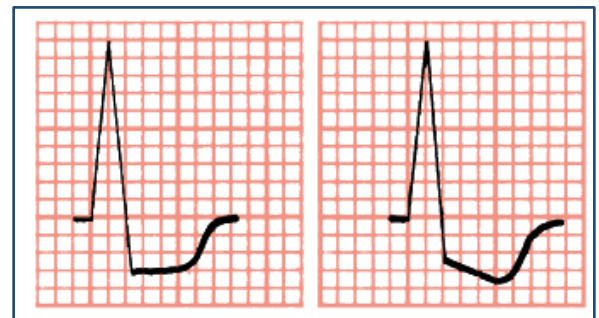
The appearance of T wave inversions in leads V_1 to V_3 (with prominent S waves) is a primary abnormality that cannot be ascribed to the bundle branch block itself. The problem of diagnosing infarction with LBBB is further complicated by the fact that the LBBB pattern has several features that resemble those seen with infarction. Thus an LBBB pattern can mimic an infarct pattern. LBBB typically shows slow R wave progression in the chest leads because of the reversed way the ventricular septum is activated (i.e., from right to left, the opposite of what happens normally). Consequently, with LBBB a loss of the normal septal R waves is seen in the right chest leads. This loss of normal R wave progression simulates the pattern seen with an anterior wall infarct. In this case, anterior wall infarction was not present. Notice that the ST segment elevations in the right chest leads resemble the pattern seen during the hyperacute or acute phase of an infarction. ST segment elevation in the right chest leads is also commonly seen with LBBB in the absence of infarction.

As a general rule, a patient with an LBBB pattern should not be diagnosed as having had an MI simply on the basis of poor R wave progression in the right chest leads or ST elevations in those leads. However, the presence of Q waves as part of QR complexes in the left chest leads (V_5 and V_6) with LBBB generally indicates an underlying MI. In addition, the appearance of ST segment elevations in the left chest leads or in other leads with prominent R waves suggests ischemia, as do ST segment depressions in the right leads or other leads with an rS or a QS morphology.

The subendocardium is particularly vulnerable to ischemia because it is most distant from the coronary blood supply and closest to the high pressure of the ventricular cavity. This inner layer of the ventricle can become ischemic while the outer layer (epicardium) remains normally perfused with blood.

The most common ECG change with subendocardial ischemia is ST segment depression. The ST depression may be limited to the anterior leads (I, aVL, and V_1 to V_6) or to the inferior leads (II, III, and aVF), or it may be seen more diffusely in both groups of leads.

The ST segment depression with subendocardial ischemia has a characteristic squared-off shape. (ST segment elevation is usually seen in lead aVR.)



Acute transmural ischemia produces ST segment elevation, a current of injury pattern. This results from epicardial injury. With pure subendocardial ischemia, just the opposite occurs; that is, the ECG shows ST segment depression (except in lead aVR, which often shows ST elevation).

In summary, myocardial ischemia involving primarily the subendocardium usually produces ST segment depression, whereas acute ischemia involving the epicardium usually produces ST elevation.

Angina The term angina pectoris refers to transient attacks of chest discomfort caused by myocardial ischemia. Angina is a symptom of coronary artery disease. The classic attack of angina is experienced as a dull, burning, or boring sub sternal pressure or heaviness. It is typically precipitated by exertion, stress, exposure to cold, and other factors, and it is relieved by rest and nitroglycerin.

Many (but not all) patients with classic angina have an ECG pattern of subendocardial ischemia, with ST segment depressions seen during an attack. When the pain disappears, the ST segments generally return to the baseline.

The ECGs of some patients with angina do not show ST depressions during chest pain. Consequently, the presence of a normal ECG does not rule out underlying coronary artery disease. However, the appearance of transient ST depressions in the ECG of a patient with chest pain is a very strong indicator of myocardial ischemia.

If ischemia to the subendocardial region is severe enough, actual infarction may occur. In such cases the ECG may show more persistent ST depressions instead of the transient depressions seen with reversible subendocardial ischemia.

Is it possible for Q waves to appear with pure subendocardial infarction?

The answer is that if only the inner half of the myocardium is infarcted, abnormal Q waves usually do not appear. Subendocardial infarction generally affects ventricular repolarization (ST-T complex) and not depolarization (QRS complex). However, important exceptions may occur, and so-called nontransmural infarctions, particularly larger ones, may be associated with Q waves.

Another ECG pattern sometimes seen in non-Q wave infarction is T wave inversions with or without ST segment depressions. (T wave inversions may also be seen in some cases of non infarctional ischemia.)

In summary, non-Q wave infarction can be associated with either persistent ST depressions or T wave inversions.

Myocardial ischemia clearly can produce a wide variety of ECG changes. For example, infarction may cause abnormal Q waves in association with ST segment elevations followed by T wave inversions. Subendocardial ischemia (e.g., during an anginal attack or a stress test) may produce transient ST depressions. In other cases, infarction may be associated with ST depressions or T wave inversions without Q waves.

In brief

- Area of necrosis >> pathological Q
- Tissue damage >> elevated ST segment
- Ischemia >> inverted T wave or peaked T

Notes ☺

- Presence of pathological Q >> old myocardial infarction
- Finger print of MI >> is the pathological Q
- Elevated ST segment with pathological Q >> recent Myocardial infarction

3. Heart Block

Some people are born with heart block (congenital), while others develop it during their lifetimes (acquired).

Acquired heart block is more common than congenital heart block. The three types of heart block are :

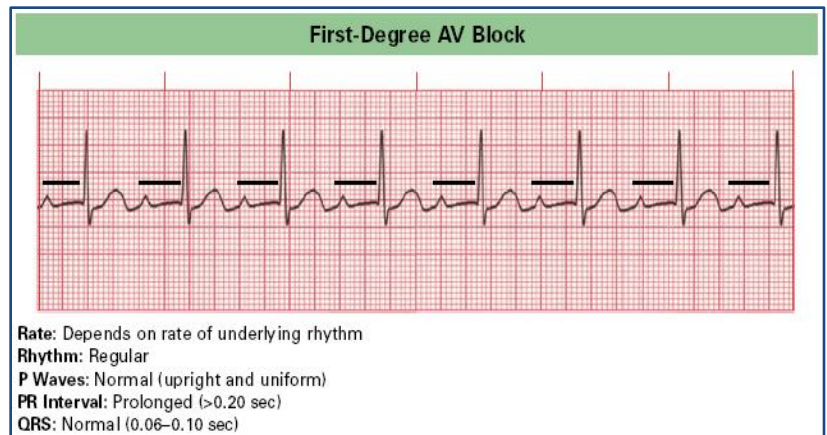
- ✚ first degree heart block
- ✚ second degree heart block
- ✚ third degree heart block

First degree is the least severe, and third degree is the most severe. This is true for both congenital and acquired heart block.

First degree heart block

The heart's electrical signals are slowed as they move from the atria to the ventricles. All signals reach from the atria to the ventricles.

Just prolonged PR interval



How to differentiate between first degree heart block and sinus bradycardia ?

In sinus bradycardia normal ECG with low rate, while in first degree heart block just prolonged PR interval.

First-degree heart block rarely causes any symptoms, and it usually doesn't require treatment.

Second degree heart block

In this type of heart block, electrical signals between the atria and ventricles are slowed to a large degree. Some signals don't reach the ventricles. On an ECG, the pattern of QRS waves doesn't follow each P wave as it normally would.

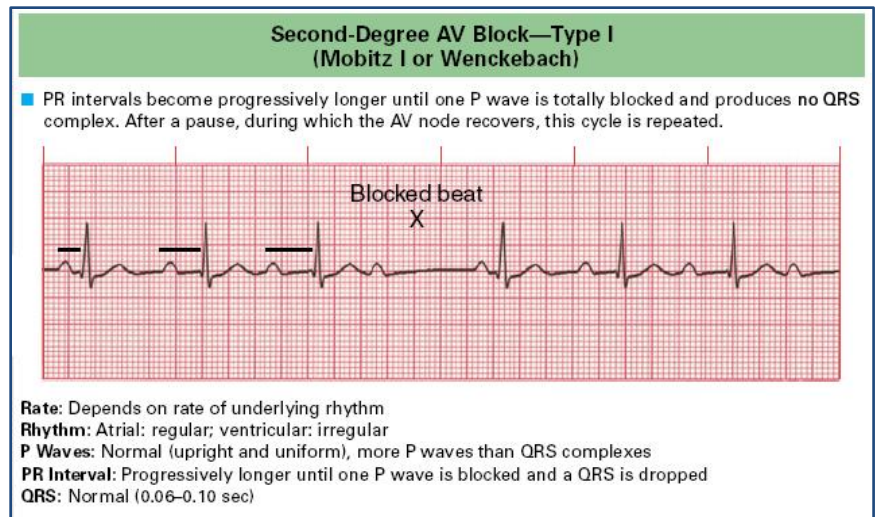
If an electrical signal is blocked before it reaches the ventricles, they won't contract and pump blood to the lungs and the rest of the body.

Second-degree heart block is divided into two types:

- Mobitz one
- Mobitz two

Mobitz one

In this type (also known as Wenckebach's block), the electrical signals are delayed more and more with each heartbeat, until the heart skips a beat. On the EKG, the delay is shown as a line (called the PR interval) between the P and QRS waves. The line gets longer and longer until the QRS waves don't follow the next P wave. Sometimes people who have Mobitz type I feel dizzy or have other symptoms. This type of second-degree heart block is less serious than Mobitz type II.



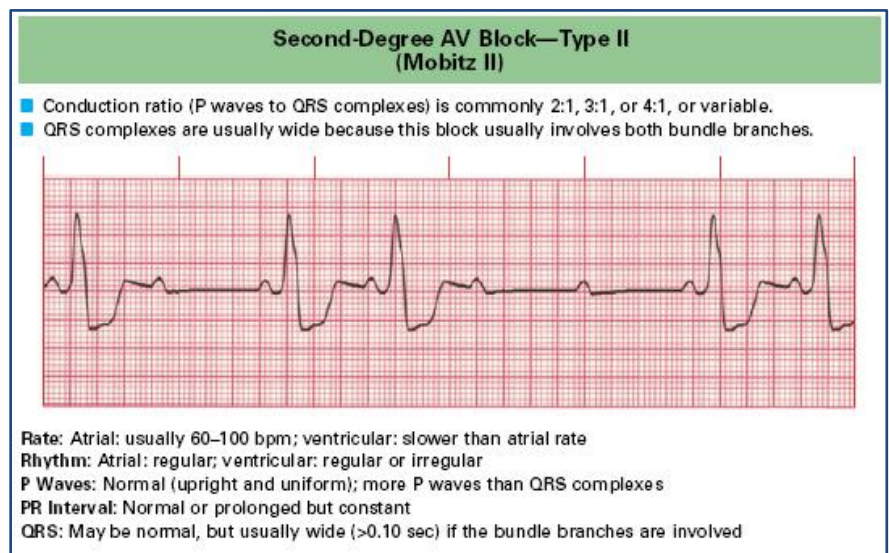
Mobitz Two

In second-degree Mobitz type II heart block, some of the electrical signals don't reach the ventricles. However, the pattern is less regular than it is in Mobitz type I.

Some signals move between the atria and ventricles normally, while others are blocked.

On an EKG, the QRS wave follows the P wave at a normal speed. Sometimes, though, the QRS wave is missing (when a signal is blocked).

Mobitz type II is less common than type I, but it's usually more severe. Some people who have type II need medical devices called pacemakers to maintain their heart rates.



Third degree heart block

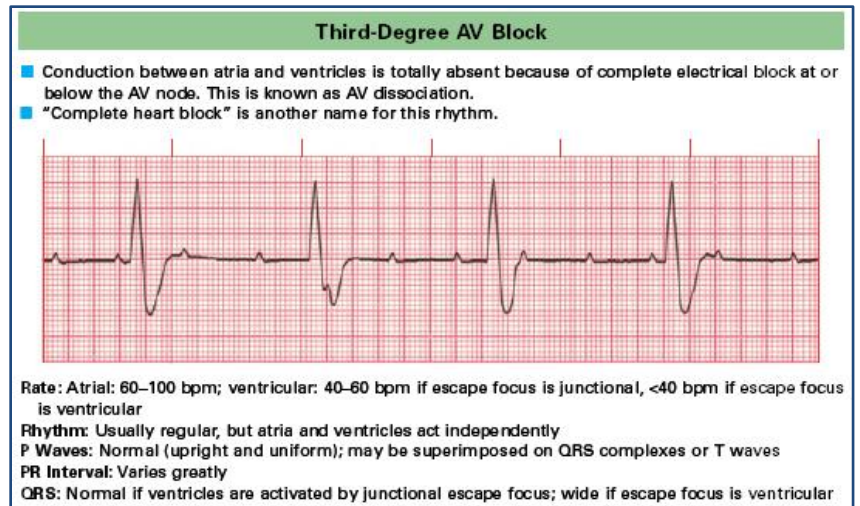
In this type of heart block, none of the electrical signals reach the ventricles. This type also is called complete heart block or complete AV block.

When complete heart block occurs, special areas in the ventricles may create electrical signals to cause the ventricles to contract.

This natural backup system is slower than the normal heart rate and isn't coordinated with the contraction of the atria.

On an EKG, the normal pattern is disrupted. The P waves occur at a faster rate that isn't coordinated with the QRS waves.

Complete heart block can result in sudden cardiac arrest and death. This type of heart block often requires emergency treatment. A temporary pacemaker may be used to keep the heart beating until you get a long-term pacemaker.



Notes

- All type of heart block are regular except>> Mobitz one
- All types of heart block with normal QRS complex except >> third degree heart block

4. Others

ECG as a Clue to Acute Life-Threatening Conditions without primary Heart or Lung Disease

- Cerebrovascular accident (especially intracranial bleed)
- Drug toxicity
 - ✓ Tricyclic antidepressant overdose, digitalis excess, etc.
- Electrolyte disorders
 - ✓ Hypokalemia
 - ✓ Hyperkalemia
 - ✓ Hypocalcemia
 - ✓ Hypercalcemia
- Endocrine disorders
 - ✓ Hypothyroidism
 - ✓ Hyperthyroidism
- Hypothermia

How to interpret an ECG

When you see an ECG paper

Relax and take a deep breath ☺

Don't forget to comment on ten points

1. Rhythm

- ✓ Sinus or not ??
- ✓ Regular or not ??

2. Rate

- If regular rhythm >> the heart rate equates $300 / n$ (which n the number of big squares between RR interval).
- Or $1500 / n$ (which n the number of small squares between RR interval), more accurate.
- If irregular rhythm >> count the number of R waves in 30 big squares and multiply the result by 10.
- Or, $300 / n$ (which n the average number of RR interval)

3. Axis

- ✓ Lead one and two (aVF) positive >> normal axis
- ✓ Lead one (positive) and lead two (aVF) negative >> left axis deviation
- ✓ Lead one (negative) and lead two (aVF) positive >> right axis deviation

4. P wave

Normally >> 2.5 X 2.5 small squares

- ✓ > 2.5 small squares (tall) and peaked >> right atrial strain
- ✓ > 2.5 small squares (width) broad >> left atrial strain

5. P-R interval

From the beginning of the P wave to the beginning of the complex, measuring 3 to 5 small squares

6. QRS complex

- ✓ Q wave >> first negative wave in the complex
- ✓ R wave >> first positive wave in the complex
- ✓ S wave >> the negative wave following R

Q wave >> less than 1 small square width and less than one fourth of the next R

R wave >> between 2 and 3 small squares width and between 1 to 5 big squares tall

S wave >> has a special relation with R wave

7. ST segment

From the end of S wave to the beginning of T wave (important in MI)

8. T wave

Never absent, less than 6 small squares width and less than one third of the preceding R (tall)

How to diagnose an ECG

First look at the rhythm, it may be :

- Regular
- Irregular (better)

Irregular rhythm

Why irregular rhythm is better ?

As they are usually one of three

- ✓ Atrial fibrillation
- ✓ Extra systole
- ✓ Mobitz one

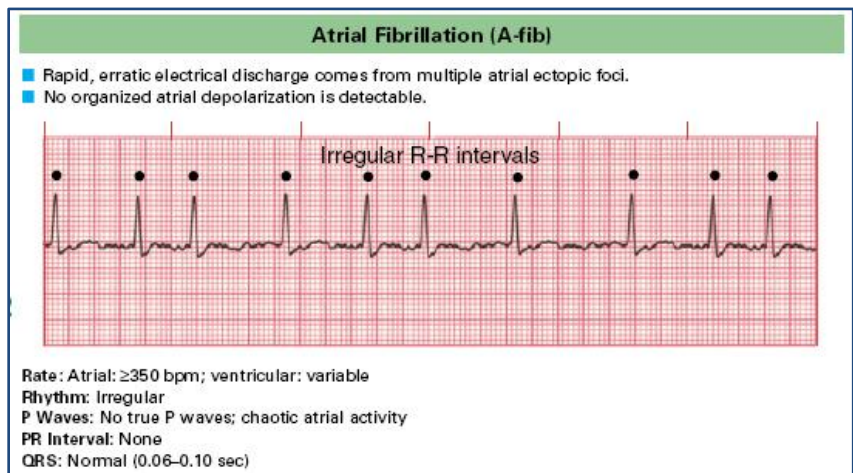
Atrial fibrillation

How to differentiate ?

- ✚ Irregular
- ✚ Usually tachycardia
- ✚ Absent P wave

AF is usually rapid, but Slow AF in certain cases :

- Patient on digitalis
- Patient on Beta blocker
- Associated with Heart block
- Lone AF

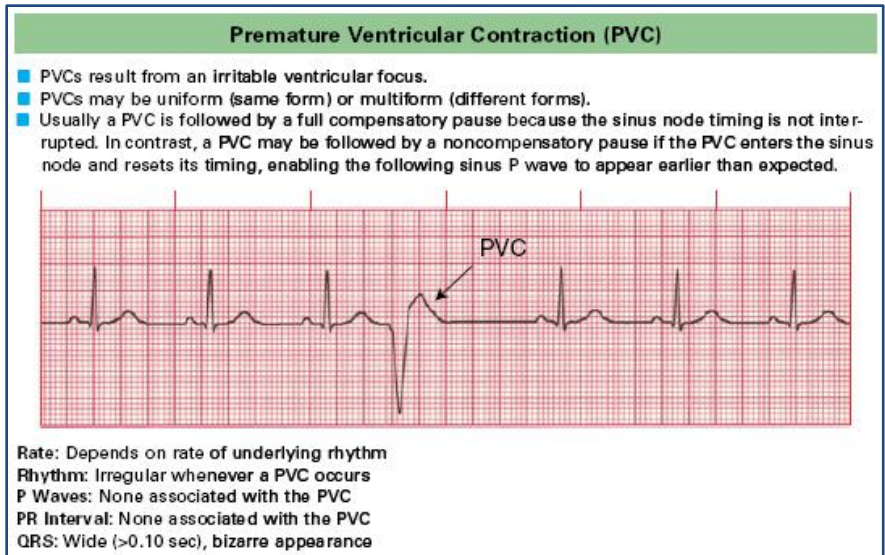


Irregular ECG with absent P wave >> AF however, tachy or not

Extra systole

Extrasystoles are essentially extra beats, or contractions, which interrupt the normal regular rhythm of the heart.

They occur when there is electrical discharge from somewhere in the heart other than the SA node. They are classified as atrial or ventricular extrasystoles (VEs) according to their site of origin.



Extrasystoles can occur frequently in people with completely normal hearts and often do not cause any problems. However, they can also be a feature of certain cardiac diseases.

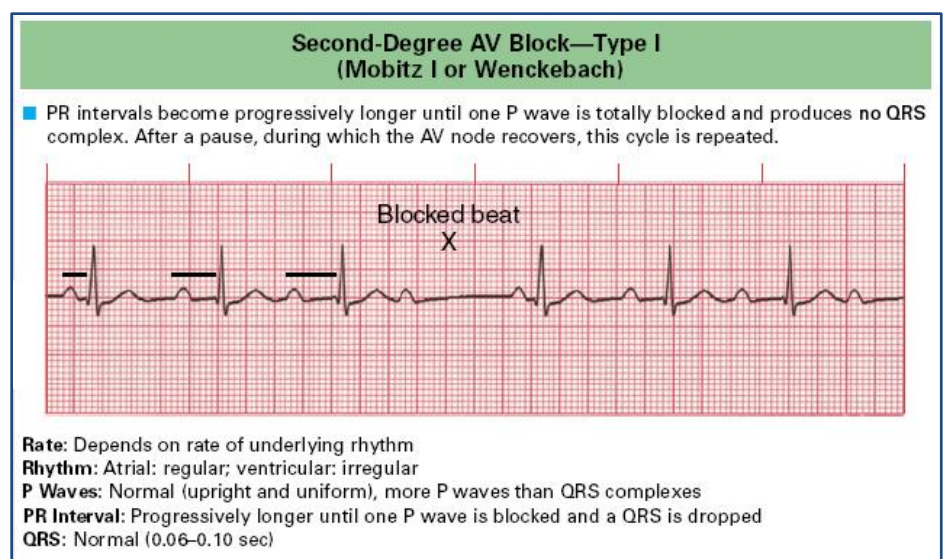
Atrial extrasystoles are premature P waves which look different from a normal P wave. They may be hidden in the ST segment or T wave of the preceding sinus beat. They may be followed either by a normal QRS complex, or the PR interval may be prolonged, or the impulse may not be conducted at all.

Ventricular extrasystoles (VEs) are wide, abnormally-shaped QRS complexes. Extrasystoles occurring at every second or third beat are called bigeminy or trigeminy respectively.

Mobitz one

How to differentiate it ?

Progressive prolongation of PR interval until dropped QRS



Regular rhythm

Look at the rate :

- Tachycardia
- Bradycardia

Normocardia as tachycardia 😊

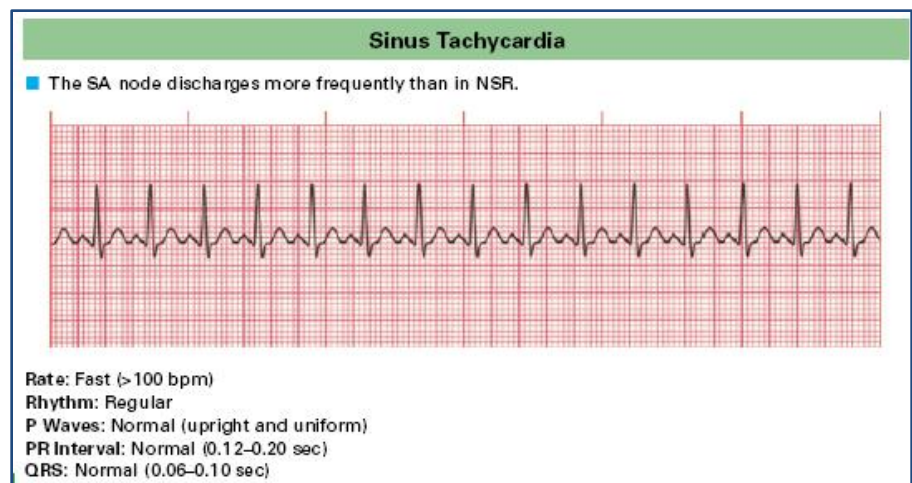
Regular tachycardia

- ✓ Sinus tachycardia
- ✓ Ventricular tachycardia
- ✓ Supra ventricular tachycardia
- ✓ Atrial flutter

Sinus Tachycardia

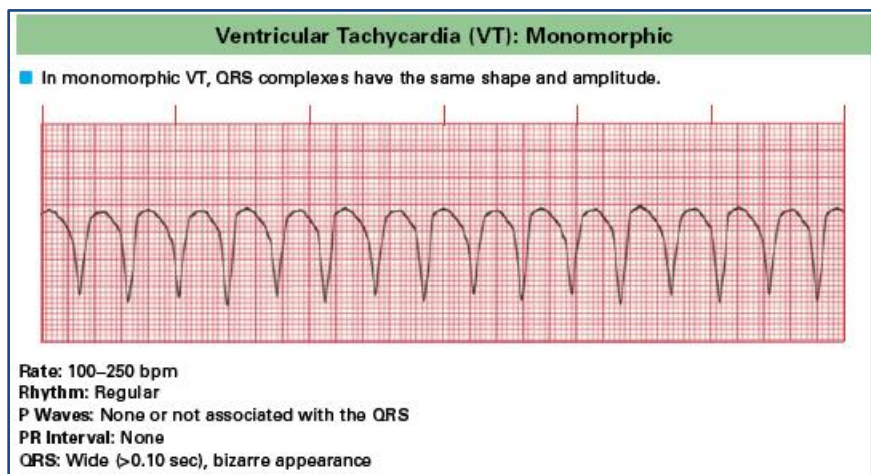
Sinus tachycardia is a rhythm in which the rate of impulses arising from the sinoatrial (SA) node is elevated.

Each sinus P wave is followed by a QRS complex, indicating sinus rhythm with 1:1 AV conduction.



Ventricular tachycardia

Ventricular tachycardia is defined as a sequence of three or more ventricular beats. The frequency must be higher than 100 bpm, mostly it is 110-250 bpm. Ventricular tachycardias often originate around old scar tissue in the heart, e.g. after myocardial infarction.



Also electrolyte disturbances and ischemia can cause ventricular tachycardias. The cardiac output is often strongly reduced during VT resulting in hypotension and loss of consciousness. VT is a medical emergency as it can deteriorate into Ventricular fibrillation and thus mechanical cardiac arrest.

Ventricular tachycardia can be catechORIZED as follows:

- ✚ **Non-sustained VT:** three or more ventricular beats with a maximal duration of 30 seconds.
- ✚ **Sustained VT:** a VT of more than 30 seconds duration (or less if treated by electrocardioversion within 30 seconds).
- ✚ **Monomorphic VT:** all ventricular beats have the same configuration.
- ✚ **Polymorphic VT:** the ventricular beats have a changing configuration. The RR interval is 180-600 ms (comparable to a heart rate of 100-333 bpm).
- ✚ **Biphasic VT:** a ventricular tachycardia with a QRS complex that alternates from beat to beat. Associated with digoxin intoxication and long QT syndrome.

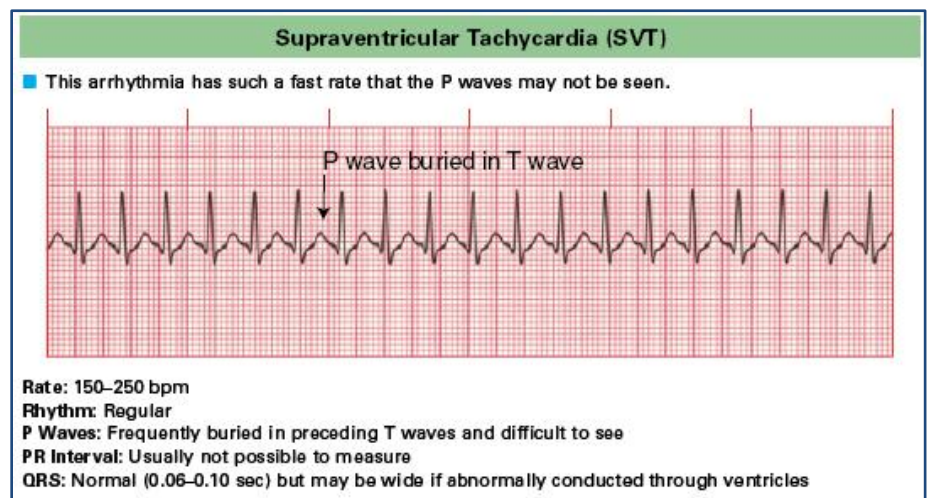
Supra ventricular tachycardia

Is a condition presenting as a rapid heart rhythm originating at or above the AV node.

It may be originating from :

- Atrium
- A.V. node

If it is originated from the atrium the P wave will be deformed. (reversal of electric current)



If it is originated from the ventricle the P wave may (inverted or absent or deformed)

Note

- *Supra ventricular tachycardia :*
May be associated with absent P, inverted P or deformed P
- *Although "SVT" can be due to any supraventricular cause, the term is most often used to refer to a specific example, paroxysmal supraventricular tachycardia (PSVT)*

Atrial flutter

Is an abnormal heart rhythm that occurs in the atria of the heart. AV node makes reduction of the atrial beats in a mathematical fashion (AV node transmit one of 2 or 3 or 4 beats)

Has a characteristic feature of Sawtooth appearance

What is the differences between atrial flutter and atrial fibrillation ?

- Atrial flutter >> is regular
- Atrial fibrillation >> is irregular

Summary

I have a regular long strip, I found there is tachycardia :

1. Look at the QRS :
 - deformed
 - Narrow normal

If deformed >> ventricular tachycardia

If narrow normal >>

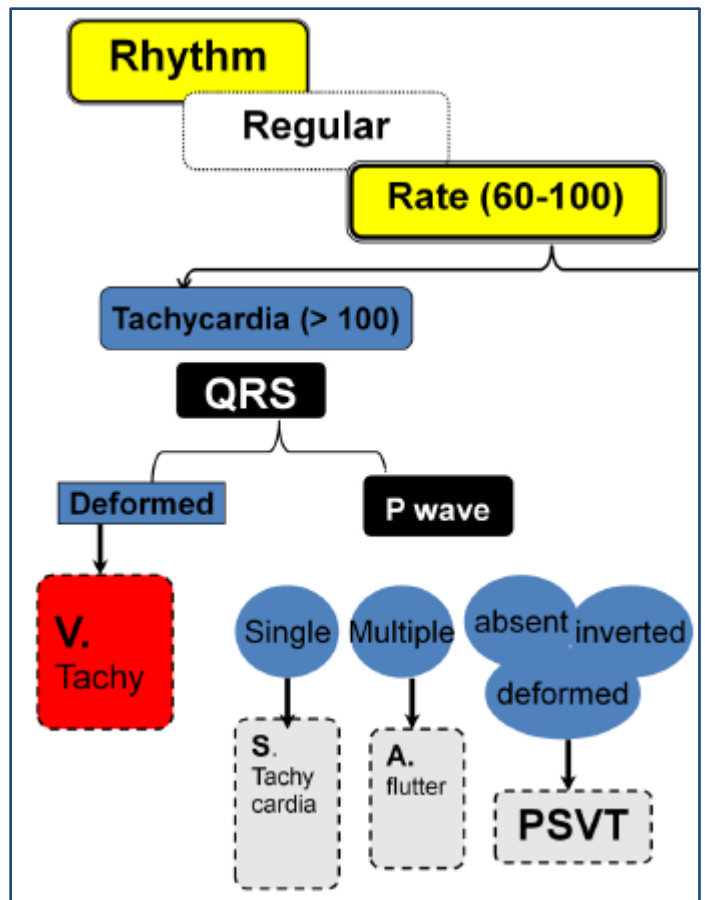
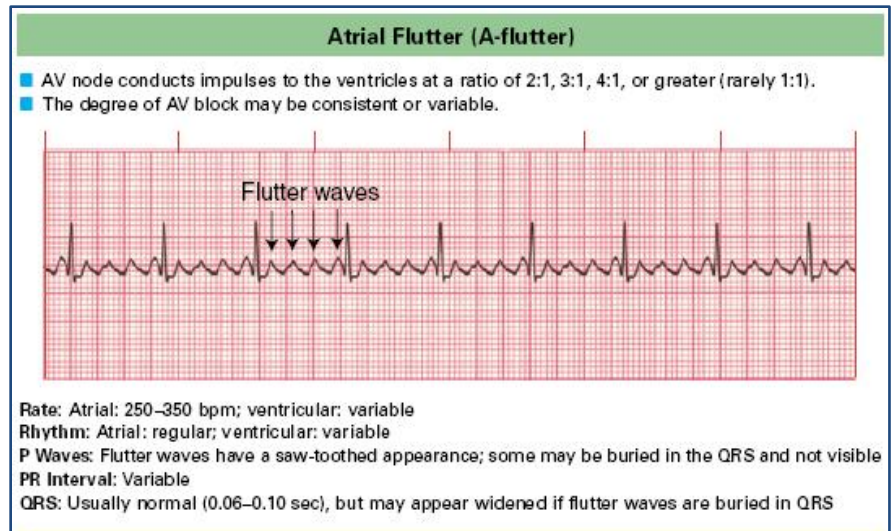
Look at P wave

- single
- multiple
- Others

Single >> sinus tachycardia

Multiple (sawtooth) >> atrial flutter

Others >> supra ventricular tachycardia

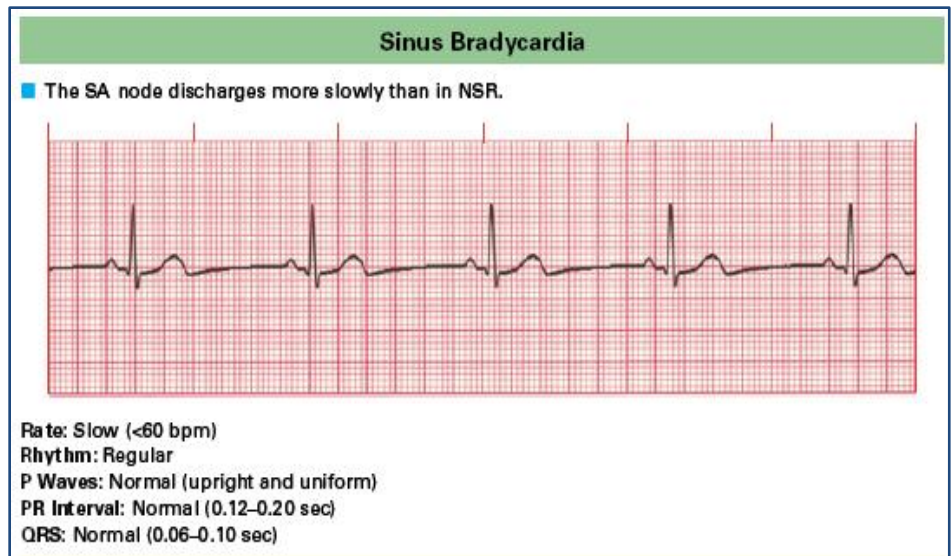


Regular bradycardia

- ✓ Sinus bradycardia
- ✓ first degree heart block
- ✓ Mobitz two
- ✓ third degree heart block
- ✓ Nodal rhythm

Sinus bradycardia

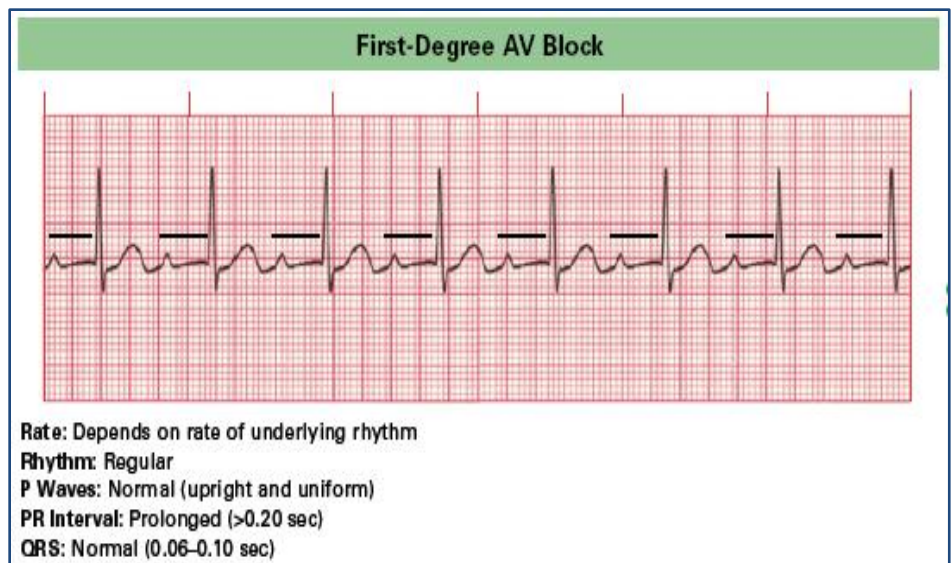
Is a heart rhythm that originates from the sinus node and has a rate of under 60 beats per minute.



First degree heart block

What is meant by first degree heart block ???

Just prolonged PR interval



Mobitz two

What is meant by Mobitz two ?

Regular drop of QRS complex

Second-Degree AV Block—Type II (Mobitz II)

- Conduction ratio (P waves to QRS complexes) is commonly 2:1, 3:1, or 4:1, or variable.
- QRS complexes are usually wide because this block usually involves both bundle branches.



Rate: Atrial: usually 60–100 bpm; ventricular: slower than atrial rate
Rhythm: Atrial: regular; ventricular: regular or irregular
P Waves: Normal (upright and uniform); more P waves than QRS complexes
PR Interval: Normal or prolonged but constant
QRS: May be normal, but usually wide (>0.10 sec) if the bundle branches are involved

Third degree heart blockWhat is meant by 3rd degree heart block ?

Deformed QRS with AV dissociation

Third-Degree AV Block

- Conduction between atria and ventricles is totally absent because of complete electrical block at or below the AV node. This is known as AV dissociation.
- "Complete heart block" is another name for this rhythm.

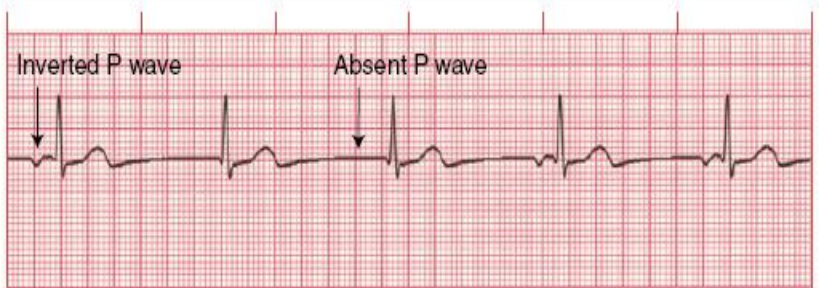


Rate: Atrial: 60–100 bpm; ventricular: 40–60 bpm if escape focus is junctional, <40 bpm if escape focus is ventricular
Rhythm: Usually regular, but atria and ventricles act independently
P Waves: Normal (upright and uniform); may be superimposed on QRS complexes or T waves
PR Interval: Varies greatly
QRS: Normal if ventricles are activated by junctional escape focus; wide if escape focus is ventricular

Nodal rhythm

What is meant by nodal rhythm ?

The AV node is the peace maker of the heart, hence the P wave will be inverted or absent

Junctional Rhythm

Rate: 40–60 bpm
Rhythm: Regular
P Waves: Absent, inverted, buried, or retrograde
PR Interval: None, short, or retrograde
QRS: Normal (0.06–0.10 sec)

Summary

I have a regular long strip, I found there is bradycardia :

1. Look at the QRS :

- Deformed
- Narrow normal

If deformed >> third degree heart block

If narrow normal >>

Look at P wave

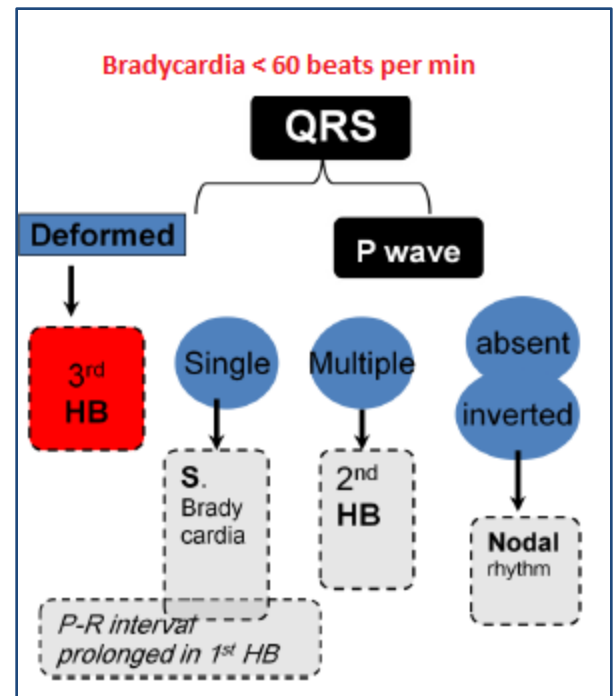
- Single
- Multiple
- Others

Single >>

- Sinus bradycardia (normal ECG with heart rate below 60 beats per minute)
- First degree heart block (just prolonged PR interval)

Multiple >> Mobitz two

Others >> Nodal rhythm



How to differentiate between atrial flutter and Mobitz two ?

- ❖ Mobitz two >> bradycardia
- ❖ Atrial flutter >> tachycardia